TROPICAL DISEASES BUREAU.

TROPICAL VETERINARY BULLETIN.

VOL. 2.] 1914. [No. 4.

BABESIASIS (PIROPLASMOSIS) AND ANAPLASMOSIS.

(317) LAVERAN (A.) & FRANCHINI (G.). **Contribution à l'Etude des "Marginal Points" des Hématies des Mammifères.** [Contribution to the Study of "Marginal Points" in the Blood Corpuscles of Mammals.]—*Bull. Soc. Path. Exot.* 1914. July. Vol. 7. No. 7. pp. 580–584.

This paper contains a brief resumé of the references in literature to marginal points and to bodies having a close morphological resemblance to them.

The authors' observations have been carried out upon the blood of young animals of different species, and they have been able to find bodies having the morphological characters of marginal points in a number of them.

The blood of ten white mice weighing from 2 to 10 grammes was examined. In four of these anaplasma-like bodies, which the authors definitely describe as anaplasms, were not rare, and in the remainder they were present in small numbers. They were found in very small numbers in the blood of one of three adult white mice.

The authors have also discovered similar bodies in young grey mice and white rats, and in the blood of young rabbits and guinea-pigs, moles, cats, dogs, calves, goats, pigs, donkeys, and monkeys.

The morphology and staining reactions of the bodies are briefly described.

The authors conclude that bodies morphologically identical with those described by THEILER occur in the blood of young mammals apart from any infection, and that it does not appear to be possible to consider these as being of a parasitic nature. But it is said that, from the observations of SMITH and KILBORNE, and a number of other authors, it is possible that anaplasma-like bodies may represent a stage in the development of certain piroplasms.

(C107) Wt.P.11/47 1,200 12.14. B.&F.Ltd.

(318) Carpano (M.). **Le Recidive nella Piroplasmosi. Su di un Tipico Caso di Recidiva nell'Asino.** [Relapses in Piroplasmosis. A Typical Case of Relapse in the Donkey.]—*La Clinica Veterinaria.* 1914. July 15. Vol. 37. No. 13. pp. 535–542. With 1 chart and 1 text fig.

In some of his previous papers the author has called special attention to the presence of latent forms of the parasite which occur in the blood of animals, and especially horses, affected with piroplasmosis, these forms having the morphological characters of anaplasms. They become active when for any reason the vitality and resistance of the host is lowered. A donkey which failed to become infected when inoculated subcutaneously with blood containing a small number of *Babesia caballi*, and also when subsequently inoculated intravenously with blood containing large numbers of *Nuttallia equi*, and was therefore immune to these parasites, had a relapse following an intravenous injection of streptococcic endotoxin derived from a very virulent strain.

Parasites of the Nuttallia type were found in the blood on the 12th and 13th days after the inoculation, and there was some amount of systemic disturbance.

The animal had been at the laboratory for 14 months before the injection which gave rise to the relapse was administered, and during that time it had not been exposed to infection by means of ticks.

(319) Martinez (I. G.). **Canine Babesiasis in Porto Rico.**—*Jl. Trop. Med. & Hyg.* 1914. July 1. Vol. 17. No. 13. p. 194.

Martinez briefly records the discovery of *Babesia canis* in the blood of dogs during the routine examinations for epizootic diseases and for rabies.

(320) Goodall (A.). **The Trypanblue Treatment in Piroplasmosis of Domesticated Animals in South Africa.**—*Parasitology.* 1914. May. Vol. 7. No. 1. pp. 62–68.

The notes contained in this paper are the outcome of the writer's experience of the use of the drug under practical conditions in country districts.

The solution used in every case was a 2 per cent. solution, made up with rain water, filtered and sterilised.

For dogs the dose used varied from 5 to 30 cc. according to the patient's weight, and was given subcutaneously.

Horses and large mules received 200 cc.; donkeys, ponies and small mules, 150 cc.; and cattle, 50 to 250 cc. In each of the latter cases the drug was given intravenously.

Good results have been obtained with all the species mentioned, and details are given of a few cases in each species. Conclusions:—

"1. Parasites are not always present in the peripheral blood of dogs, and are often extremely difficult to find in other animals, especially under conditions encountered in the field. If well-marked clinical symptoms are shown one is justified in using Trypanblue, even if parasites cannot be satisfactorily demonstrated.

BRITISH MUSEUM

"2. A large dose of a weak solution of Trypanblue should be administered. The writer cannot help thinking that the failures attributed to the drug by other observers are due, in many cases, to too small a quantity of the solution being injected.

"3. After an experience of the treatment in some hundreds of cases, the writer feels justified in stating that animals do not suffer from relapses if a sufficient quantity of the solution is used.

"4. The dog can become infected a second time after Trypanblue treatment, but this second infection is also amenable to the drug."

TRYPANOSOMIASIS.

(321) Bruce (D.), Hamerton (A. E.), Watson (D. P.) & Lady Bruce. **Trypanosome Diseases of Domestic Stock in Nyasaland. III.**—*Trypanosoma pecorum.* **Development in** *Glossina morsitans.*—*Proc. Roy. Soc.* 1914. Aug. 6. Series B. Vol. 88. No. B 600. pp. 33–37. With 1 coloured plate.

In a previous paper the authors have described the morphology and pathogenic range of this trypanosome, and in the present paper an account is given of its development in *G. morsitans.*

The trypanosome belongs to the group which does not invade the salivary glands. The development first takes place in the gut, and then extends forwards to the proboscis and hypopharynx, where the development into "blood forms" occurs.

Seven transmission experiments were carried out, and of these five were positive. Two of these were at laboratory temperature, and in the remainder the flies were kept in an incubator. Ten per cent. of the flies used were found to be infected.

From the details given it appears that a period of from 19 to 53 days may elapse before flies become infective.

In the positive experiments the majority of the flies showed infection of the proboscis, and all showed infection of one or more portions of the intestine. The salivary glands were always negative.

In the negative experiments there was infection of the labial cavity of one only. One or more parts of the intestine showed infection in every case, but the salivary glands were uniformly negative.

The coloured plate shows the cycle of developmental forms of *T. pecorum* in *G. morsitans.* In the intestine long parasites which are indistinguishable from the corresponding forms of other pathogenic parasites occur. When attached to the labrum the crithidial form is assumed, and in the proboscis the "blood forms," which are the only infective form, are developed.

(322) Bruce (D.), Hamerton (A. E.), Watson (D. P.) & Lady Bruce. *Glossina brevipalpis* **as a Carrier of Trypanosome Disease in Nyasaland.**—*Proc. Roy. Soc.* 1914. Aug. 6. Series B. Vol 88. No. B 600. pp. 20–32. With 1 coloured plate.

G. brevipalpis is found in a tract of country estimated at about 5 square miles at the mouth of the Lingadzi river on the west shore of Lake Nyasa. This area is traversed by two main roads, one running

east and west and the other parallel to the shore and about one mile west of it.

During the day no specimens of the fly are ever seen, but as evening approaches specimens may be seen sitting motionless in the road. They do not settle upon or follow passers-by, and would take no notice of a dog repeatedly walked through their haunts towards evening. They do not move about in search of food, nor do they chase each other.

Five hundred flies were caught and every one was a male. Some of them were found on dissection to contain mammalian blood. When kept in captivity they remain dormant on the sides of their cages during the day, but become active at night. If the side of the cage be placed up against a dog or a goat they will feed with avidity at any time during the day or night.

Examinations made to ascertain the natural food shewed that of 59 flies examined seven contained blood. In six cases it was probably that of antelope and in the other human.

Of the several hundred flies dissected, only four were found to be females, and it would appear that while the males are found on the paths the females remain in the jungle. The same kind of thing was observed to a lesser extent with *G. morsitans*.

Dissections of wild flies shewed that 44 out of 496 were infected with trypanosomes.

While trypanosomes were found in the proboscis, and in one instance in the salivary glands, the great bulk of them occurred in the mid and fore-gut, and in the smaller number of cases in the hind gut.

It is stated that in 19 cases it was possible to make a more or less correct guess at the identity of the trypanosomes found in the flies. These are *T. brucei* vel *rhodesiense*, 1; *T. pecorum*, 9; *T. simiae*, 1; and *T. grayi*, 8. In 10 flies the flagellates were considered to belong to a pathogenic type of unknown species, and in the remainder no opinion could be expressed.

Five experiments were made with flies brought from the lake shore; in each case a monkey, a dog, and a goat being used for feeding experiments. In only one case was the result positive, *T. pecorum* being found.

A number of experiments were carried out to ascertain whether *G. brevipalpis* is capable of acting as a carrier of various pathogenic trypanosomes.

T. brucei vel *rhodesiense*.—Wild flies were used in the experiments, as it was found to be impossible to breed flies for the purpose, both on account of the distance that they had to be carried from the lake to the laboratory (50 miles), and on account of the scarcity of the flies.

Wild flies were fed upon animals infected with the trypanosome causing disease in man in Nyasaland and then upon healthy animals. In six experiments 232 flies were used, but the result was negative in every instance. Fifty-three flies were dissected and eight infected flies were found. In one of these there was infection of the proboscis and alimentary tract, in one infection of the salivary gland and of the alimentary tract, and in the remainder infection of the aimentary tract only. The two flies in which there was infection other than

that of the intestine were found in one batch. That in which the salivary glands were found to be infected was dissected at the 33rd day, and the other at the 46th day. The former of these had the salivary glands swarming with trypanosomes, and, although inoculation of a rat failed, it is thought probable that it represented a true development of the Nyasaland trypanosome, the development not having reached the infective stage.

In a coloured plate are shewn the morphological details of the trypanosomes occurring in the fly.

In the intestine of the fly long slender forms are found, and these invade the salivary glands. Here the parasite acquires a crithidial form, in which the posterior extremity is attenuated while the anterior end becomes broader. Later the posterior portion becomes short again and the parasite is short and thick. The parasite then becomes rounded in form, and masses of them in this condition are found in the lumen of the glands. These masses break up into individuals in which the micronucleus is again posterior and the parasite gradually assumes the fully developed trypanosome, or "blood form." It is pointed out that although wild flies were used, this is the only type of trypanosome which invades the salivary glands of the flies in the district and that therefore *T. pecorum*, *T. simiae*, and *T. caprae* are excluded.

T. brucei Zululand, 1913.—Three experiments were carried out, using 200 flies. One was positive and two negative.

Only 12 flies were dissected, and in one long trypanosomes apparently of the pathogenic type were found in the intestine, but it was impossible to identify them. A dog became infected 58 days after the flies had been fed upon an infected dog. Only two flies of this batch were dissected, and both were negative. It is probable that the dog was infected by a fly in which development of *T. brucei*, Zululand, had taken place, as if there had been a fly in the cage naturally infected with the Nyasaland strain, the dog ought to have shewn trypanosomes earlier than the 58th day.

Further experiments of the same kind shewed that *G. brevipalpis* is capable of acting as a carrier for both *T. pecorum* and *T. caprae*. The flies dissected in these experiments shewed trypanosomes in the proboscis and intestine, but not in the salivary glands.

(323) Chalmers (A. J.) & O'Farrell (W. R.). **Sleeping Sickness in the Lado of the Anglo-Egyptian Sudan.**—*Jl. Trop. Med. & Hyg.* 1914. Sept. 15. Vol. 17. No. 18. pp. 273–284. With 1 map and 5 charts.

In this paper are compared *T. rhodesiense* and the Yei strain of trypanosomes. The former was brought from Liverpool and the latter was obtained from patients at Yei in the Lado district of the Mongalla Province.

The experimental work was done at Khartoum owing to the possibility of introducing *T. rhodesiense* into the sleeping sickness area. The patients infected with the Yei strain were not brought to Khartoum for examination because of the danger of spreading the disease, and because it was not desired to interfere with their treatment.

All the blood films were fixed wet with osmic acid vapour for about four seconds and then transferred to absolute alcohol, in which they

were kept for two to five minutes. Giemsa's stain was used in two dilutions: either 1 drop to 1 cc. with the addition of a drop or two of 1 per 1,000 potassium carbonate, in which they were stained for an hour or longer, or 2·5 cc. to 100 with 5 drops of carbonate solution, in which they were stained for 5 to 24 hours.

A summary of the state of affairs regarding African human trypanosomes and an interesting account of the history of the occurrence of sleeping sickness in the countries adjoining the Lado are given.

From these it appears that there are two main areas of infection in the Mongalla Province, namely a western area centered round Yei, and an eastern area adjoining Kajo-Kaji.

The Yei strain was obtained from a monkey inoculated by RANKEN from a patient at the Sleeping Sickness Segregation Camp at Yei.

It would appear that these two districts obtained their infection originally from two different sources. The first appears to have been infected from the Belgian Congo and possibly through the medium of Baganda traders. The source of the infection of the second district appears to have been Uganda.

The infected animals, two monkeys and a dog, were inoculated from an untreated case.

The authors state that the strain may represent the strain present in Uganda or that on the Congo, but they consider this to be of little importance as they believe that the Uganda infection was originally derived from the Congo.

Morphology.—The parasite varied in length from 18 to 36 microns, and in width from 1 to 2·5 microns. The average length of 1,000 trypanosomes, measured 100 daily, in the blood of an infected monkey was 25 microns. Measuring was commenced on the first day that the animal shewed a heavy infection, viz., the 10th, and it was continued to the 19th day. The monkey died on the 33rd day. A posterior-nuclear form was never observed.

Animal experimentation shewed that the virulence of the trypanosome for dogs, cats, rabbits, gerbils, jerboas, white rats, and monkeys, is less than that of *T. rhodesiense*, but greater than that of *T. nigeriense* Scott-Macfie.

In the dog the period of incubation was 12 days, and the animal often lived several months. In the monkey the period of incubation was nine days, and the duration of life averaged about 35 days. In the gerbil the period of incubation was 7 days, and the duration of life 14.

A dog was rendered immune, i.e., its peripheral blood failed to shew trypanosomes for more than 60 days after its fifth inoculation with the Yei strain. The last tested gerbil inoculated with the blood of this dog failed to become infected.

The serum of this dog destroyed the Yei trypanosome in vitro in 20 minutes, but was without effect upon *T. rhodesiense* after an hour.

At a time when the immunity was incomplete the serum was mixed with the different strains of trypanosomes and immediately inoculated into gerbils. In the case of the Yei strain an infection resulted, and the animal was recovering from it when it died of heat stroke along with 49 other animals. This occurred on the 39th day. The *rhodesiense* mixture resulted in an infection which proved fatal on the 7th day.

When the immunity was complete the serum was mixed with infected blood in the proportion of 10 to 1, and after 30 minutes was injected into gerbils. No trypanosomes were seen in the blood of the gerbil inoculated with the Yei strain, but it was killed accidently on the 13th day after inoculation. No trypanosomes were found in the internal organs, but smears from the lungs shewed perculiar bodies contained in cells which resembled the granules found in the spleens of patients and of animals inoculated with kala azar. With *T. rhodesiense* the gerbil died of infection on the 10th day.

Cross immunity is to be dealt with in a subsequent paper.

Cytolytic sera.—Human serum was not found to have any trypanolytic effect upon either of the human strains of trypanosomes or upon two strains of mule trypanosomes, but it did appear to have a distinctly beneficial effect upon the Yei strain, as after an hour the trypanosomes were more active than at the commencement of the observation.

It is recorded that a strong though incomplete agglutination was observed to be produced by a normal human serum upon a mule strain of trypanosomes, and it is pointed out that if this is admitted agglutination is useless as a specific test.

No experiments have been carried out regarding the transmission of the trypanosomes.

A detailed comparison is made with each of the other human trypanosomes, excluding the original strain of DUTTON, 1902, which the authors have not been able to trace.

The conclusions arrived at may be summarised as follows :—

The strain sent in by RANKEN and the Congo strain are the same, and probably they and the Uganda strains are identical. There being no data of which the authors are aware to compare these strains with *T. gambiense*, Dutton, 1902, they think that provisionally it would be safer to keep the name *T. castellanii*, Kruse, 1903, for these strains until more light is thrown upon the question as to what is *T. gambiense*, Dutton, 1902.

It appears that sleeping sickness in Africa can be divided into three categories.

(1) Southern sleeping sickness caused by *T. rhodesiense* and spread by *G. morsitans*.

(2) Equatorial sleeping sickness caused by *T. castellanii*, Kruse, 1903, and spread by *G. palpalis*.

(3) Northern sleeping sickness which may be caused by as yet imperfectly known trypanosomes, named :—

(*a*) *T. gambiense*, Dutton, 1902.

(*b*) *T. nigeriense*, Scott-Macfie, 1913, and possibly other trypanosomes.

The authors believe that BRUCE's method of charting trypanosomes and LAVERAN and MESNIL's method of cross-immunity testing are both of distinct value in the comparison of parasites.

The question as to why some cases of the disease in the Lado Enclave are exceedingly chronic while others are very acute may be answered in two ways. The more probable reason is that the chronic cases occur in the area that has been longest infected, while the acute cases occur in the area which has recently become infected.

The less likely answer is that there are two forms of the disease.

(324) MESNIL. (F.) & RINGENBACH (J.). **Sur le *Trypanosoma rhodesiense* et ses Affinités avec le *Tr. gambiense*. Quatrième Note.** [*Trypanosoma rhodesiense* and its Affinities with *T. gambiense*. Fourth Note.]—*Bull. Soc. Path. Exot.* 1914. July. Vol. 7. No. 7. pp. 612-618.

The authors refer to a paper published by them in 1911 in which it was recorded that a monkey which had a strong immunity to two strains of *T. gambiense* subsequently became infected with *T. rhodesiense* although the period of incubation and the duration of life were longer than in monkeys having no immunity to *T. gambiense*.

Reference is also made to LAVERAN'S experiment in which a goat immune to *T. gambiense* contracted a fatal infection which lasted only 35 days when inoculated with *T. rhodesiense*.

The conclusion is drawn from these results and from those of MESNIL and LEGER, in which mice inoculated with *T. rhodesiense* and cured with arsenophenylglycin were tested as to their susceptibility to *T. gambiense*, that the two viruses constitute distinct but closely related species.

The present paper records experiments carried out with a strain of *T. rhodesiense* (Case 16, W. G.) obtained from WENYON, and the strain originally studied by the authors in 1911–12.

In the opinion of the authors, negative results obtained with cross immunity experiments are less conclusive than positive ones.

In the rat WENYON found that the duration of life varied from 12 to 39 days, but in the series of passages through the rat carried out by the authors the period ranged from five to 18 days only.

In the mouse the course of the infection is still more variable than in the rat. The duration of life varied from four to 25 days, and in cases in which the animals survived for more than 15 days crises occurred during which trypanosomes were not discoverable in the blood by microscopical examination. In the course of the later passages the duration of life was longer than in the earlier ones.

A similar variability of effect was also observed in the guinea-pig.

The authors' own strain, on the other hand, was markedly constant in its effects. Mice were regularly killed in four days.

Guinea-pigs died in 28 and 22 days, as compared with 19 and 79 days with WENYON'S strain.

A series of experiments was carried out with four goats, two of which were immune to *T. gambiense* and two susceptible. Each of the strains of *T. rhodesiense* was used to inoculate an immune and a susceptible goat.

Goat B. immune to *T. gambiense*.—Inoculated with the authors' strain of *T. rhodesiense* became infected and died. Trypanosomes appeared in the blood in small numbers a month after inoculation, and they were observed on a number of occasions afterwards, but apparently always in small numbers. The goat died six weeks after the appearance of trypanosomes in the blood. There was oedema of the face and simple keratitis. Duration of life, 68 days.

Goat B[1]. Non-immune to *T. gambiense*.—Infection resulted, but trypanosomes were not numerous in the blood. There was slight oedema of the face, but there is no mention of keratitis. The animal died in 42 days.

Goat C. Immune to *T. gambiense*.—Inoculated with WENYON's strain. Although a single trypanosome was seen in the blood on one occasion only about seven weeks after inoculation, the infectivity of the blood was repeatedly proved by the inoculation of mice. The animal died on the 54th day.

Goat C^{1}.—Non-immune goat inoculated with WENYON's strain of *T. rhodesiense*. Microscopic examination of the blood was negative throughout, but its infectivity was proved by the inoculation of mice from time to time. The animal died on the 61st day.

It is concluded from these experiments that the evidence tends to show that *T. rhodesiense* and *T. gambiense* are distinct.

A few serum experiments were carried out, and it was found that the sera of the two non-immune goats possessed no activity against *T. gambiense*. The sera of Goats B and B^{1} were found to be active against *T. rhodesiense* of the authors' strain, but to be practically without effect upon WENYON's strain. The sera of Goats C and C^{1} were found to be fairly active against WENYON's strain, but very slightly against the authors' strain.

In 1911 the authors stated that their strain of *T. rhodesiense* behaved like an animal trypanosome of the nagana type from the point of view of the action of human serum upon it. WENYON's strain was found to have practically the same property.

(325) BRUCE (D.), HAMERTON (A. E.), WATSON (D. P.) & Lady BRUCE. **Infectivity of *Glossina morsitans* in Nyasaland during 1912 and 1913.**—*Proc. Roy. Soc.* 1914. Aug. 6. Series B. Vol. 88. No. B 600. pp. 43–48.

During 1912, 1,975 flies were caught and dissected. Of these 86 per cent. were males and 14 per cent. females. 6·53 per cent. were found to be infected with trypanosomes. In 1913, 1,060 flies were dissected, and 8·58 were found to be infected.

During 1912, 60 attempts were made to determine the infectivity of the flies by injecting the intestinal contents into animals, but only three inoculations were positive. This was no doubt to a large extent due to the fact that the intestinal forms are not infective, a point subsequently discovered. In only two cases were the salivary glands found to be infected. Inoculation of these glands into rats confirmed the diagnosis that the infection was *T. brucei* vel *rhodesiense*.

In 1913, an attempt was made to diagnose directly the species of trypanosome with which the fly were infected, and no injections were made.

Invasion of the salivary glands could only be *T. brucei* vel *rhodesiense*; invasion of the intestine, labial cavity and hypopharynx meant *T. pecorum* or *T. simiae*, and size would distinguish between these. Lastly, if only the labial cavity and hypopharynx were seen to contain flagellates, *T. caprae* was indicated.

T. brucei vel *rhodesiense* was found once, *T. pecorum* six times, *T. simiae* 12 times, and *T. caprae* 14 times.

(326) Bruce (D.), Hamerton (A. E.), Watson (D. P.) & Lady Bruce. **Trypanosomes found in Wild *Glossina moritans* and Wild Game in the "Fly-Belt" of the Upper Shiré Valley.**—*Proc. Roy. Soc.* 1914. Aug. 6. Series B. Vol. 88. No. B 600. pp. 38–41.

The "fly-belt" in question extends along the Upper Shiré River Valley from Lake Pamalombe to the Murchison cataracts. It is about 100 miles south of Kasu. It is separated from the "fly" area on the west shore of Lake Nyasa by a range of hills where fly is absent, but which forms no obstacle to the passage of infected animals from one area to the other.

Game is abundant in the district, particularly in the dry season. Cases of trypanosomiasis have been observed in man, and the natives are unable to keep cattle. Their goats and dogs are constantly being destroyed by trypanosomiasis.

Seven experiments were carried out, but only dogs and goats were used, as there were no monkeys available. The different batches of flies, which numbered from 73 to 650 in a batch, were allowed to feed upon a dog and a goat twice.

Six of the dogs became infected with *Trypanosoma brucei* vel *rhodesiense*. Two of these were at the same time infected with *T. pecorum*, and one contracted a *pecorum* infection alone. These were the only trypanosomes found in the dogs.

Six of the goats became infected with *T. pecorum*, one of these also had *T. simiae*, four were infected with *T. caprae*, and one showed a *caprae* infection only.

These experiments show that the fly in the Upper Shiré district carries the same species of trypanosomes as those found in the flies from the Proclaimed Area.

A zebra, 7 impala, 2 koodoo, and 6 waterbuck were examined for trypanosomes, the examination involving the microscopic examination of blood preparations. *T. brucei* vel *rhodesiense* was found in one waterbuck, *T. pecorum* was found in 2 impala and 2 waterbuck, *T. caprae* was found in an impala, a koodoo, and a waterbuck, and *T. ingens* was found in one waterbuck.

(327) Bruce (D.), Hamerton (A. E.), Watson (D. P.) & Lady Bruce. **The Food of *Glossina morsitans*.**—*Proc. Roy. Soc.* 1914. Aug. 6. Series B. Vol. 88. No. B 600. pp. 41–42.

Five hundred flies, freshly caught in the Proclaimed Area, were killed by chloroform and a smear made from the intestine of each. These were fixed with osmic acid and alcohol, stained with Giemsa, and examined for the presence of blood corpuscles.

The proportion of male to females was roughly two to one.

Of the 500 flies examined, 57·6 per cent. were found to contain mammalian blood in a recognisable state. No measurements of the corpuscles were made owing to changes in the corpuscles induced by digestion, but cells of the small type such as are seen in the blood of antelope predominated.

In three cases nucleated red corpuscles were found, and in one of these only were there no mammalian corpuscles present at the same time. Measurement of these corpuscles showed that they were in all

probability avian and not reptilian. No vegetable matter was found in any of the flies.

From experiments carried out with laboratory flies it appeared that blood was not recognisable after the third day. Hence it may be inferred roughly that half the flies examined had fed within three days, and that therefore flies naturally feed at least once in six days.

Trypanosomes were recognised in 2·8 per cent. of the flies, and it is probable that a higher percentage was actually infected.

(328) Owen (G. E.). **Mechanical Transmission of Trypanosomiasis.**—*Jl. Comp. Path. & Therap.* 1914. Sept. Vol. 27. No. 3. pp. 259–260.

The author's object in writing this paper is to point out the possibility of mechanical transmission becoming serious under suitable conditions, rather than to lay stress upon its frequency and importance.

During the period 1908–1913, cattle to the number of 2,500-3,000 died from trypanosomiasis in the Barotse reserve of Northern Rhodesia, although the district is free from *Glossina morsitans*. Special interest attaches to the fact that the mortality began each year about the beginning of February and ceased after June. The wet season, when flies are most numerous, lasts from December to March.

There were in the district some dozen horses, some being bred every year, and large numbers of sheep, dogs and goats, but no natural case of trypanosomaisis had ever been encountered among these animals. The explanation of this fact did not lie in the trypanosome being pathogenic for cattle only, as the author found all the other species susceptible to it, and particularly the dogs.

In seeking an explanation of these facts the first point of importance is that the trypanosomiasis was definitely confined to certain herds, others grazing over the same ground being free, and in every native herd there was a history of exchanging cattle with, or hiring to traders and transport riders.

In 1908, an owner came into the district with sick cattle. He lost the majority of them from trypanosomiasis in three years, and with his advent the disease became very noticeable.

In 1912, when the writer went to investigate, he at once resorted to isolation, with the result that in 1913 he had over 800 head of cattle without loss in an area which had been the centre of the disease the previous year. These animals remained out of a lot of 1,100. They were carefully watched for about six months after the fly season, all infected and suspected animals being removed before the flies became numerous again.

During April and May, 1912, the author saw 200 cases, whereas from February to May inclusive 1913 only five cases were seen, all occurring in a suspected herd.

Had a fly or a tick been associated in the transmission, the disease could not have remained confined to certain herds while others on the same veld remained unaffected.

Tabanidae, Haematopota and Stomoxys are the commonest flies in the district.

During the months when the flies are most numerous and during the hot period of the day, the cattle crowd together for mutual pro-

tection. To watch a herd crowded together and see the Tabanidae swarming on them is sufficient to impress one with the fact that the conditions are very favourable for mechanical transmission.

The following experiment was made :—

Two kraals were erected within 10 yards of each other. In one two healthy cows and a heifer were placed, and in the other two healthy cows and two infected cows. The cattle from the two kraals were grazed over the same ground, but never at the same time, and the kraals were occasionally changed.

The two healthy cattle in the second kraal became infected, but the healthy animals in the other kraal remained uninfected. The object of exchanging the kraals was to ascertain whether mosquitoes in the kraals would act as transmitters.

Dogs, sheep, and goats were exposed to infection by mixing healthy with clean, but no infection resulted.

It was noticed that while dogs allowed Stomoxys to feed upon them freely, they bit at any Tabanidae coming near them. No positive results were obtained with experiments carried out to test cyclical or mechanical transmission with either of these species.

The author came to the conclusion that the trypanosome, which was not identified, but was responsible for a chronic infection in the cattle, was transmitted in a mechanical manner by Tabanidae.

(329) Cross (H. E.). **Experiments on the Treatment of Surra in Camels.**—*Memoirs Dept. Agric., India.* Veterinary Series. 1914. Sept. Vol. 2. No. 6. pp. 155–198. With 2 figs.

This paper contains an account of the experiments carried out with the following six methods of treatment :—*

1. One dose of soamin followed by gradually increasing doses of arsenious acid ; the treatment being commenced when the trypanosomes were present in the peripheral blood.
2. As above, but treatment started when the trypanosomes were absent from the circulation.
3. Gradually increasing doses of arsenious acid ; treatment being started when the trypanosomes were present in the blood.
4. As No. 3, but started when trypanosomes were absent from the peripheral blood.
5. Combination of soamin, tartar emetic, and arsenious acid ; treatment being started when trypanosomes were present in the blood.
6. Soamin and arsenious acid given alternately, with one day interval between each ; treatment being started when trypanosomes were present in the blood.

As a preliminary investigation a number of experiments were made to ascertain the minimum lethal dose of arsenious acid when administered intravenously.

---

*Reference to the list of treatments given at the commencement of the paper appears to indicate that there has been a little confusion in the numbering of treatments 3 and 4, as in the detailed statements of the experiments under these numbers the factor regarding the presence or absence of trypanosomes at the time when treatment was started appears to have been reversed. The numbering given in the original has been followed in this abstract.

The tolerance of the camel for arsenious acid is very variable and consequently each case has to be treated upon its merits to some extent. It was found, however, that as a general rule 1·2 grammes per 1,000 lb. body weight can be safely given, but in some cases larger doses (1·7 gm.) were tolerated.

The arsenious acid was used in every case in a 1 per cent. solution dissolved in alkali. Tartar emetic was also administered in a 1 per cent. solution. The solutions were filtered and injected at 37° C. by means of Ehrlich's injection flasks.

In treating cases of colic large doses of chlorodyne (3 to 4 ounces), containing 1 grain of hydrochloride of morphia to the ounce, should be administered. In cases of unsteadiness 2 grains of strychnine should be given subcutaneously.

A liberal diet should be allowed during and after treatment.

Treatment 1.—The dose of soamin used was 100 cc. of a 5 per cent. solution and the dose of arsenic ranged from 0·4 gm. to, in some cases, over 2 gm., about 10 doses of arsenic being given, and one day elapsing between consecutive doses.

In all, 21 animals were treated. In 12 cases there were relapses, in two cases the animals died, and in the remaining seven cases there was no relapse recorded for periods varying from 111 to 281 days. The percentage of animals in which no relapse was recorded was 33.

Treatment 2.—Nine animals were subjected to this method of treatment. In two cases there were relapses, in two instances the animals died, and in the remaining five no relapses were recorded for periods varying from 95 to 281 days.

In this case 55 per cent. of the animals did not show any relapses.

Treatment 3.—Arsenious acid alone was administered in doses increasing from 0·4 gm. to 2 gm. or more, there being, as before, an interval of a day between consecutive doses.

The following table summarises the results obtained by this method :

Number of animals treated	..	12
Cases in which relapses occurred		4
Number of Deaths	.. ..	1
Cases in which no relapse occurred		7 (122–277 days under observation).

Treatment 4. The same as No. 3, but started when trypanosomes were present in the blood.—

Eight animals were treated by this method. In four cases there were relapses, and in the remaining four no relapses were recorded for periods ranging from 121–178 days.

Treatment 5. Combined soamin, tartar emetic, and arsenious acid.—

These drugs were given on three consecutive days and then repeated three times with intervals of two to six days. The doses of soamin and emetic was 100 cc. of a 5 per cent. solution (and in a few cases 6 or 7 per cent.) and 0·5 gm. respectively. The dose of arsenic was gradually increased from 0·5 to 1·2 gm.

The tabulated results are as follows :—

Animals treated	.. .. ..	24
Cases in which relapses occurred		9
Number of Deaths	.. ..	1
Cases in which no relapse occurred		14 (73 to 379 days under observation.)

Percentage of cases in which no relapses were recorded—58 per cent.

Treatment 6. Soamin and arsenious acid given alternately.—The soamin was given in a 5 to 7 per cent. solution, and the arsenious acid in doses increasing up to a maximum of 1·5 grammes. Included among these experiments are some in which the treatment was continued by arsenious acid alone after some doses of soamin had been administered.

Fifteen animals were included in this group. In four cases there were relapses. In one death occurred, and in the remaining 10 (66 per cent.) no relapses were recorded in from 80 to 279 days.

The conclusions drawn may be summarised as follows :—

There is no necessity to wait for the appearance of trypanosomes in the circulation before commencing treatment. This is of importance because sometimes the intervals between paroxysms last for weeks and time is thus saved.

No case should be considered as cured until it has been under observation for a year without a relapse occurring.

Soamin should not be used in solutions stronger than 5 per cent. as there is a danger of producing nephritis. While cures can be effected without increasing the doses of arsenic to the subtoxic dose, the dose should be increased till the sub-tonic dose is reached, the principle of the treatment being the sterilisation of the tissues.

In the combined treatment, the dose of arsenic must not be so large as when it is given alone or when alternated with soamin.

As the tolerance of the camel to arsenic varies from animal to animal and the virulence of the trypanosome varied in different outbreaks, the same percentage of cures cannot always be obtained.

Good feeding is essential.

(330) Ciuca (M.). **L'Action de quelques Substances Médicamenteuses sur le Pouvoir Alexique du Sérum.** [The Effect of Certain Medicinal Substances upon the Alexic Power of Serum.]—*Bull. Soc. Path. Exot.* 1914. July. Vol. 7. No. 7. pp. 626–632.

The experiments detailed in this paper were carried out during the course of an investigation into the mechanism of the action of emetic upon trypanosomes.

Reference is made to the observations of Fenyvessy and Freund, who found that while the intravenous injection of calcium chloride increased the alexic power of the serum, the opposite effect was produced when the drug was added to serum in vitro.

The observations of Weil and Dufourt and of Weisback are also referred to. These authors found respectively that the intravenous injection of 2 to 4 cc. of a 4 per cent. solution of bicarbonate of soda causes an increase in the alexic power of the serum, both in man and in rabbits, and that salvarsan produces an accelerating effect upon haemolysis in vitro and particularly upon the alexin.

So was unable to detect any influence upon the alexin when rabbits were injected subcutaneously with atoxyl.

The author's experiments have been carried out upon rabbits, guinea-pigs, and rats, both normal and infected with the Uganda strain of Nagana

The experiments were carried out in the following way :

The rabbits and guinea-pigs were injected intravenously and the rats intraperitoneally with emetic in the proportion of 1 to 5 mgm. per 100 gm. body weight, atoxyl in doses up to 2 mgm. per 100 gm. and salvarsan 1·5 mgm. per 100 grammes.

The animals were either bled to death half an hour afterwards or they were bled on a number of occasions at variable intervals. The results of the experiments are set out in a number of tables.

It was found that the intravenous injection of emetic or salvarsan into healthy animals caused an elevation of the alexic power, while atoxyl produced the opposite results. The latter effect was far more marked than the opposite effect produced by emetic and salvarsan. That the effect was due to an action upon the alexin is shown by the fact that heating all the sera to 56° C. inactivated them. The effect could be observed for 48 hours after the injection.

With regard to animals severely affected with trypanosomiasis, it was confirmed that at the height of infection alexin is generally quite absent from the serum, but in these animals when treated with emetic or salvarsan the increase in the amount of alexin was particularly marked. Such sera were able to activate systems which could not be activated by sera from non-infected animals three hours after the treatment.

Atoxyl decreases the alexin in non-infected animals, but increases it in infected animals, but to not so great an extent as the other two drugs.

Among healthy rabbits and rats the alexin is sometimes particularly susceptible to the action of chemical salts.

In the earlier experiments the control serum was taken from an animal before the injection of the drug. The inconclusive and sometimes inconstant results were due to a diminution in the amount of alexin following on the previous bleeding.

In some of the experiments the alexin was found to have fallen to a quarter of the normal titre when an animal was bled twice on the same day or on two consecutive days.

The increase in the alexin is associated with and follows the intense leucocytosis produced by the injection of the drugs.

The fall in the amount of alexin produced in healthy animals by atoxyl is probably due to the affinity of this drug for the albumens of the body, this being essential to render it active (Levaditi and Yamanouchi). It is quite possible that in animals infected with trypanosomes the product which is formed in the body has an action comparable to that provoked by salvarsan and emetic.

(331) Laveran (A.) & Roudsky (D.). **Sur un Dérivé du Diamino-arsénobenzène.** [A Derivative of Diaminoarsenobenzene.]—*Bull. Soc. Path. Exot.* 1914. July. Vol. 7. No. 7. pp. 593–596.

The substance described in this paper has been prepared by Oechslin and has been named O 1. It is said to possess the following advantages over arsenophenylglycin, which it closely resembles in its action in trypanosome diseases.—

The drug is far more stable. It occurs in the form of a pale yellow powder which is easily soluble in water. It is far cheaper to prepare and does not undergo rapid alterations when sealed up in tubes, even

when the tubes contain a small amount of air. A watery solution is said to remain unchanged in sealed ampoules for periods varying from eight days to a month. It is less toxic than arsenophenylglycin A healthy mouse weighing 20 grammes will withstand a dose of 10 mgm. whereas a dose of 8 mgm. of arsenophenylglycin would suffice to produce a fatal effect. As animals infected with trypanosomes are more susceptible to the action of drugs than healthy ones, the dose for an infected mouse must not exceed 5 mgm. For a guinea-pig weighing 500 grammes (whether infected or not is not stated) the dose is 5 cgm., and for a dog weighing 10 kgm., 25 cgm.

No danger attaches to repeated treatments with O 1. Sub cutaneous injections produce painful oedematous swellings, and sometimes gangrene of the skin, but a 4 per cent. solution in distilled water has been injected into the saphena vein of dogs without ill effects.

Sterilisation in the autoclave increases the toxicity of the drug, and the authors have resorted to filtration for the sterilisation of solutions destined for intravenous injection.

The subcutaneous injection of 5 mgm. into mice heavily infected with *T. brucei*, *T. rhodesiense*, or *T. dimorphon* has caused the disappearance of the trypanosomes within a few hours. Cures have been effected by this treatment and also by three injections of 2 to 3 mgm.

In the dog and the guinea-pig it is easy to cause a disappearance of trypanosomes from the circulation, but the authors are not in a position to state whether relapses occur, and in what proportion of cases.

A cure has been effected in the case of a guinea-pig infected with a strain of *T. gambiense* resistant to atoxyl.

Neither the composition nor the method of preparation of the drug is given.

(332) Levaditi (C.) & Mutermilch (St.). **Amboceptеurs et Arsénobenzol.** [Amboceptors and Arsenobenzol.]—*Bull. Soc. Path. Exot.* 1914. July. Vol. 7. No. 7. pp. 633–640.

The authors have prepared three kinds of sera in the following ways: (*a*) Serum from normal rabbits treated with "606." (*b*) Serum containing trypanolytic amboceptor from infected rabbits killed after the crisis. (*c*) Serum possessing the characters of the two preceding from infected rabbits which have been treated with "606" after the crisis.

These three sera were used for the treatment of infected mice.

Eight experiments have been carried out in this way, and in five of these constant results were obtained.

Comparison of the results obtained shews that what may be called the "606 alone" serum does not exercise any marked effect upon the multiplication of the trypanosomes, nor does it delay death very much. The serum containing only amboceptor exercised a very feeble effect, all the mice dying with trypanosomes in their blood. On the other hand the serum from the rabbits treated with "606" which also contained amboceptor prevented the multiplication of the parasites, the mice which received the largest doses of this serum shewing no

trypanosomes in their blood during the whole course of the experiments which lasted from 22 to 37 days.

In three of the experiments the results obtained were, however, different, in that the "606 alone" sera used in these experiments were themselves active, so that the beneficial effects of combining amboceptor with "606" were less apparent.

Having established the superiority of the sera combining the two active properties, the next step taken was to ascertain the trypanocidal action of the three sera in vitro.

The experiments were repeated several times with constant results. Two striking facts were ascertained.

(1) If the trypanocidal effect of the amboceptor + "606" complex is compared in vitro with the trypanocidal action of each of the components, the effect of the former is not found to greatly exceed the effects produced by the latter. Thus, in one instance the complex was found to be completely effective in 1 in 500. In this case the amboceptor alone was effective in the same strength and the "606 alone" was effective in 1 in 100.

From this it follows that in vitro the trypanocidal action of the amboceptor is not added to the trypanocidal action of the arsenic compound. If that were the case the complex should exceed either of the components in activity. It must therefore be concluded that amboceptor and "606" do not act upon the trypanosomes in the same manner. In EHRLICH'S terminology, the arsenic receptor is different from the receptor acted upon by the trypanolytic antibody.

(2) "606 alone" serum exercises markedly different effects in vitro and in vivo. While in the majority of cases this serum when injected almost at the same time as the trypanosomes, but at a different part of the body, did not prevent the evolution of the infection nor a fatal termination, in vitro it caused the destruction of the trypanosomes.

This difference of action can only be explained on the supposition that new properties are acquired by the "606" when it is subjected to the action of the cells and fluid of the body. It is probable that its diffusibility is altered and also its affinities for the tissues.

It is quite another matter when the arsenic compound is present in the serum in association with amboceptor. The results obtained in vitro and in vivo force one to the conclusion that in the animal body the action of one of the components of the complex assists the action of the other. Further, it appears that the two trypanocidal agents acting upon the two sets of receptors should render relapses of arsenic and antibody-resistant strains less likely and thus lead to complete sterilisation. Arsenic-fast individuals will be subjected to the toxic influence of the antibody trypanocides and vice versa. These facts may have important practical consequences.

The authors have the idea that in sera containing the "606" and amboceptor these two substances are associated with the same protein molecule, but they have not been able to get experimental results plainly indicating this. All that they have been able to shew is that the arsenical compound and the amboceptor cannot be separated by dialysis through collodion sacs, and further, that precipitation of the albumins and globulins does not lead to distinct separation. Though this suggests that the attachment may be to the same protein

molecule, a simple explanation, it is admitted that the experimental facts could be explained in another manner, such as simple absorption and physical inclusion of the arsenic compound by the precipitates.

Whatever may be the real explanation, the fact remains that the results obtained indicate a line which may have valuable practical results.

(333) Jack (R. W.). **Tsetse Fly and Big Game in Southern Rhodesia.** —*Bull. Entomol. Research.* 1914. Sept. Vol. 5. No. 2. pp. 97–110. With 2 maps.

This paper contains an account of the investigations of the author regarding the connection between *G. morsitans* and big game. During the last four and a half years he has visited the great majority of the fly-belts in the territory, and the main belts have been visited repeatedly. The broad result of the observations is that game is more or less abundant all the year round in the fly-infested country, and that in no instances is the larger animal life absent, even during a portion of the year. The evidence collected is considered to be strongly in favour of a vital association between the prevalence of big game and the continuance and increase of the fly.

The evidence in favour of the necessity of big game to the tsetse in Southern Rhodesia and the adjacent territory may be summed up under four heads:—

1. Tsetse retired before the advance of civilisation in the Transvaal, the only known modification of conditions being the destruction of game.
2. Tsetse disappeared from large tracts of country immediately after the rinderpest epizootic of 1896.
3. Tsetse has increased and spread since the rinderpest only in those parts of Southern Rhodesia where big game has increased.
4. Tsetse has greatly decreased of late years in the Hartley District in those parts where big game has been most effectively destroyed or driven away.

LEISHMANIASIS.

(334) Row (R.). **Experimental Leishmaniasis in the Monkey and the Mouse induced by the Parasites in Culture.**—*Indian Jl. Med. Research.* 1914. Apr. Vol. 1. No. 4. pp. 617–621. With 2 coloured plates.

This paper contains an account of experiments in which susceptible animals were infected with cultures of *L. donovani*, and of *L. tropica*. The principal object in recording the experiments is that they afford an easy means of testing the infectivity of this or that intervening host which may be brought forward from time to time as *the* agency responsible for the transmission of the disease to man.

A.—Kala azar culture experiments.—

The success or failure of experimental infection with cultures appears

to depend on the susceptibility of the animal used, the age and morphological phase of the parasite in culture, and the method of infection.

The smaller race of *Macacus sinicus* found in the neighbourhood of Madras is more susceptible than the larger race from the Bombay district, and this animal appears to be the subject *par excellence* for experimental purposes.

The author has not had any successes in attempting to infect dogs, although LAVERAN working with the same strain has infected three out of eight.

The author's observations have convinced him that positive results are only obtained when the cultures contain large numbers of small rounded bodies resembling the original parasites, or small coccus-like bodies which look like "fused condensed nuclei of the parasites enveloped in a mere suspicion of a zone of body protoplasm."

These results differ from those obtained by NICOLLE, MANCEAUX, LAVERAN, and GONDER, who have only obtained positive results by using young active flagellates.

Intraperitoneal inoculations if successful always produce a generalised infection, while subcutaneous inoculation leads to a localised lesion not followed by generalisation, or generalisation preceded either by a very slight localised lesion or none at all.

B. Oriental Sore culture experiments.—

The author states that, while he has been able to infect monkeys experimentally with *Leishmania tropica* from cases of Cambay sore, he has never been able to infect them with cultures of the parasite. NICOLLE and others have recorded successful inoculation of the monkey with the production of a local lesion, and GONDER has recorded infection of the spleen and liver in an inoculated mouse, with localisation at a later period.

The author has subjected a mouse to the following inoculations :—

1st injection	intraperitoneally	9–4–13, 0·5	cc.,	7 weeks	old culture.
2nd „	„	10–4–13, 0·25	cc.,	4 „	„
3rd „	„	11–4–13, 0·25	cc.,	4 „	„
4th „	„	11–6–13, 0·25	cc.,	6 „	„

All the cultures were rich in rounded forms and contained hardly any motile forms.

The animal remained alive and apparently healthy until it was killed 9 months and 13 days later.

At the post-mortem the spleen was six to eight times the normal size and the liver was also somewhat enlarged. The peritoneum and the other organs appeared normal. Smears from the liver, spleen, and bone marrow showed an exceedingly rich infection, and cultures yielded pure flagellates. In smears made from the heart blood large coccus-like bodies were found in the red corpuscles. These bodies were not cocci, as they would not grow upon agar.

These facts show that, like *Leishmania donovani*, *Leishmania tropica* in culture produces a generalised infection in the mouse, while kala azar cultures administered subcutaneously produce a local lesion, or a generalised infection, or both.

The question arises whether the parasites are identical and the author expresses the hope that further experiment will throw light upon the subject.

(335) KORKE (V. T.). **A Note on the Production of Localised Lesions by *Leishmania donovani* in *Macacus sinicus*.**—*Indian Jl. Med. Research.* 1914. Apr. Vol. 1. No. 4. pp. 622–625.

This short paper contains brief details of a number of experiments carried out to check Row's results regarding the production of localised lesions in monkeys with *Leishmania donovani*.

The conclusions arrived at are as follows:—

"1. In *Macacus sinicus*, *Leishmania donovani* is capable of producing purely localised lesions which take the form of nodules.

"2. Injecting infective material into pockets of skin on the foreheads of monkeys is an effectual and easy way of producing such lesions. Such a procedure may at times give rise to a systemic or generalised infection.

"3. The incubation period of these nodules varies between 52 and 71 days.

"4. The Leishmania contained in such nodules are capable of giving rise to similar lesions in other monkeys. They appear in typical form as well as mononuclear round bodies. Occasionally parasites in these nodules are very small though typical in other morphological respects."

(336) MACKIE (F. P.). **Kala azar in Nowgong (Assam).**—*Indian Jl. Med. Research.* 1914. Apr. Vol. 1. No. 4. pp. 626–662. With 6 maps and 6 charts.

This paper is a report of the work done during the period February to September, 1912. It is interesting to note that no connection was traced between the disease in man and its occurrence in the dog or any other species of animal.

(337) PATTON (W. S.). **The Behaviour of the Parasite of Indian Kala Azar in the Dog Flea, *Ctenocephalus felis*, Bouché, with some Remarks on Canine Kala Azar and its Relation to the Human Disease.**—*Indian Jl. Med. Research.* 1914. July. Vol. 2. No. 1. pp. 399–403.

The material examined was obtained from about 200 fleas which were fed upon a dog infected with the human parasite during the four days preceding its death. Four days before death about 500 parasites could be found in every smear of peripheral blood, and two days before death there were about 1,000 parasites in every such film. In Madras the dog flea is *Ctenocephalus felis*. *Ctenocephalus canis* is found on the jackal only.

The fleas were dissected at varying intervals after the meal of infected blood, and it was found to be exceedingly difficult to find parasites eight hours after the meal, and none were found in the fleas dissected at later intervals.

The conclusions drawn are as follows:—

"1. The parasite of Indian kala azar does not develop in the dog flea, *Ctenocephalus felis*, but degenerates and disappears in eight hours. This together with the fact that the dog has not been found infected with kala azar, or perhaps, to be exact herpetomoniasis, in India, strongly supports the view that human kala azar is not of canine origin. The human flea, *Pulex irritans*, has not been found in Madras.

"2. Assuming that the parasites of Indian and Mediterranean kala azar are identical, it is difficult to understand why the one does not develop in the flea, while the other does.

"3. The fact that the dog may be infected with *Herpetomonas ctenocephali* suggests that the so-called canine kala azar may have nothing to do with the human disease, and that its association with it is a coincidence. There is at present no proof that either *Ctenocephalus canis* or *Pulex irritans* transmits the parasite of human kala azar from dog to man and man to dog, and the hypothesis advanced by NICOLLE and so vigourously supported by BASILE and others appears to be based on very slender evidence."

(338) SERGENT (Edm.), SERGENT (Et.), LEMAIRE (G.) & SENEVET (G.) **Insecte Transmetteur et Réservoir de Virus du Clou de Biskra. Hypothèse et Expériences Préliminaires.** [Insect Transmitting Agent and Reservoir of the Virus of Oriental Boil at Biskra. Hypothesis and Preliminary Experiments.]—*Bull. Soc. Path. Exot.* 1914. July. Vol. 7. No. 7. pp. 577–579.

Epidemiological observation does not allow of any view regarding the transmission of oriental sore at Biskra other than that it is transmitted by a biting insect, and, in view of the fact that the exposed portions of the body are the predilection seats of the boils, the insect is in all probability winged.

In 1904, the authors incriminated Phlebotomus as being the transmitting agent, and since that date other authors have published papers agreeing with this view.

Since oriental boil is a localised affection (GONDER'S view that it is a generalised disease being unproved), it is scarcely possible to hold that the human subject is the reservoir whence the insects derive the virus. A reservoir must therefore be sought among the animals which are bitten by Phlebotomus.

Phlebotomus of the group *P. papatasii* bite the human subject for preference or perhaps exclusively. But those of the group *P. minutus* normally bite reptiles.

Two species are encountered at Biskra: *P. papatasii* and *P. minutus*, var. *africanus*. The Geckonidae, which have been described by HOWLETT in India as the natural host of *P. minutus*, are very commonly found in houses in Biskra, the species being *Tarentola mauritanica*.

The authors have carried out investigations with the idea of testing the hypotheses that *P. minutus* may be the transmitting agent and the tarentola the reservoir of the virus.

The seasonal occurrence of the disease may be associated with the fact mentioned by HOWLETT that *P. minutus* only bites man during the hot season, and during the cold weather lives on the hibernating geckos.

The authors have verified the statement that *P. minutus* feeds on the tarentola at Biskra, and they have also found that it bites the human subject.

At the moment their investigations permit them to say that in nearly 16 per cent. of cases cultures made from the organs of the tarentola yield pure cultures of a leptomonas resembling the leptomonas of cultures of the organism of oriental boil. Interest attaches to this point because all the cultures of leptomonas so far obtained from the organs of vertebrates have been derived from leishmania.

(339) Archives de l'Institut Pasteur de Tunis.—1914. Aug. 1. Vol. 9. No. 1. pp. 30–38.—**Chronique de Kala Azar en Tunisie.** [Statistics of Kala azar in Tunis].

Since the publication of the last statistics eight fresh cases of leishmaniasis in the human subject have been discovered in Tunis and one case in Algeria. The majority of the cases have been found in boys under three years of age.

During the year 1913, 109 dogs which presented symptoms suggestive of infection were examined, and of these six were found to be infected.

Five strains of parasites are under cultivation. Two of these are from human sources and have reached the 100th and 56th generation respectively, two are from canine cases and have passed through 61 and 58 subcultures, and the remaining strain is from a case of oriental sore which is in the 119th generation. The reinoculations are made twice monthly on blood agar. The last of the strains was isolated in 1909, and during the period that it has been under artificial cultivation it has not undergone any change of morphology.

It has been found very difficult to maintain the canine strain by passage through dogs, and it is a curious fact that the canine strain of the parasite is less infective for the dog than the human strain.

SPIROCHAETOSIS.

(340) Wolbach (S. B.). **The Distribution and Morphology of *Spirochaeta duttoni* and *Spirochaeta kochi* in experimentally infected Ticks (*Ornithodorus moubata*).—*Jl. Med. Research.*** 1914. March. Vol. 30. No. 1. pp. 37–48. With 3 plates comprising 18 figures.

The investigations described in this paper were rendered possible by the elaboration of a special method of staining with Giemsa.

The full details of the method are given and the following are the principal points :—

The tissues are fixed in sublimate alcohol and are passed through into paraffin. Sections should be 2 microns in thickness. After complete removal of the mercury by iodine, and of the iodine by hyposulphite of soda, the sections are stained with the following dilution of Giemsa : sixty drops of stain, 100 cc. of distilled water, 10 cc. of methyl alcohol and 2 drops of ·5 per cent. sodium carbonate. The stain is replaced twice during the first hour and then left acting for 20 hours.

After staining the sections are differentiated in two changes of 100 cc. of acetone containing 15 gm. of colophonium. The process takes from 15 to 30 seconds. The sections are then passed through acetone xylol, pure xylol, and mounted in cedar oil. Uniform results have been obtained.

As a result of the examination of preparations made from experimentally infected ticks treated in this way the following conclusions are drawn :—

"1. The modified Giemsa stain employed is a reliable method of demonstrating spirochaetes in the tissues of ticks.

"2. The two strains of spirochaetes studied, *Spirochaeta duttoni* and *Spirochaeta kochi*, migrate through all the organs and tissues of the ticks.

"3. Multiplication of spirochaetes does not take place in the epithelial cells of the tick, but does occur in the tissues of the connective tissue group.

"4. The minute granules and comma bodies found in epithelial cells, and probably those in other tissues, are not stages in the development of the spirochaetes.

"5. Large granules, coiled and encysted forms derived from spirochaetes, occur in various connective tissue structures, and may possibly represent resting or multiplication stages.

"6. By virtue of the power to invade tissues, the spirochaetes studied may be transmitted in any secretory or excretory product of the ticks."

(341) Todd (J. L.) & Wolbach (S. B.). **Concerning the Filterability of *Spirochaeta duttoni*.**—*Jl. Med. Research.* 1914. March. Vol. 30. No. 1. pp. 27–36.

Following the technique employed by Novy and Knapp, Breinl and Kinghorn, the authors have repeated the filtration experiments of these authors and have arrived at the following conclusions :—

"1. *Spirochaeta duttoni* in an infective form can be forced through a Berkefeld filter by pressures of over fifty pounds to the square inch.

"2. *Spirochaeta duttoni* cannot be filtered through a Berkefeld filter, in an infective form by atmospheric pressure."

MALARIAL AND OTHER BLOOD PARASITES.

(342) Phisalix (Mme). **Sur une Hémogrégarine Nouvelle, Parasite de *Boodon fuliginosus* Boïe, et ses Formes de Multiplication Endogène.** [A New Haemogregarine parasitic in *Boodon fuliginosus*, and its Endogenous Multiplication Forms.]—*Bull. Soc. Path. Exot.* 1914. July. Vol. 7. No. 7. pp. 575–577.

The snake in whose blood the parasites described in this paper were found was sent from the Soudan. It died in an extremely anaemic condition a month after its arrival.

Apart from the blood parasites the following were also found :—

On the skin *Ophionyssus natricis* and *Aponema laeve* were present, the former being in large numbers.

Numerous slender nematodes, the species of which has not yet been determined, were found in the oesophagus and in the connective tissue surrounding the viscera.

Examination of the blood showed that there was profound anaemia and that the majority of the remaining blood corpuscles had lost the greater part of their haemoglobin.

The haemogregarine was present in the peripheral and heart blood in very considerable numbers, but no multiplication forms were found in the latter. They were, however, found in the capillaries of the lungs, liver, kidneys, spleen and pancreas.

Two endoglobular forms were recognisable.

(*a.*) Vermicules measuring 14 to 15 microns in length, and 2 to 3 in width. These were slightly curved at the extremities, one of which tapered slightly. They were found applied to the nucleus of the cells, and possessed a thin unstained capsule.

The nucleus formed an elongated mass which was sometimes bilobed. It was placed close to the convex border of the parasite towards the thicker end. It was homogeneous and stained deeply. In some instances two parasites were found in a single corpuscle. The corpuscles did not appear to have undergone any alteration save an increase in size due to the presence of the parasites.

(*b.*) In a minority of cases parasites of the same length, but of three or four times the thickness, were found. In these the nucleus was rounded or annular, and composed of filaments of chromatin. These stained with moderate intensity, and in some cases there were granules scattered through the protoplasm which did not take the stain.

Free forms were represented by both of these types. In the majority of cases they were only set free by a gradual disintegration of the blood corpuscle containing them. Subsequently the parasites themselves tended to degenerate, first becoming swollen and then disintegrating. Those parasites which had previously been slender could be recognised by the slight curving of their extremities.

Cysts containing macromerozoites were encountered in small numbers in the lungs, liver, spleen, and kidneys. They were regularly ellipsoid in shape and contained 2 or 4, and rarely 6, merozoites. The smallest of these measured about 17 by 12 microns, and the largest about 30 by 20. The nuclei stained intensely, and the protoplasm very distinctly. The capsule did not stain.

Cysts containing micromerozoites were found in large numbers in the lungs, liver, and pancreas, but not in the spleen and kidneys. The youngest, corresponding to the first stages of division of the nucleus, contained 2 or 4 nuclei embedded in a granular mass. They measured about 25 by 18 microns. Those in the most advanced stage of development were packed with little ovoid nuclei measuring about 1 micron. These stained intensely. Cysts in this stage measured 30 by 25 microns. The envelope was very thin, and around a great number of them, but not all, there could be seen a retraction zone in which the parasite occupied an eccentric position.

None of the cysts were found to be in the process of rupture, and no forms of exogenous multiplication were seen.

The name *Haemogregarina boodoni* is suggested for the parasite.

(343) Acton (H. W.) & Knowles (R.). **Studies on the Halteridium Parasite of the Pigeon, *Haemoproteus columbae*, Celli and San Felice.** *Indian Jl. Med. Research.* 1914. Apr. Vol. 1. No. 4. pp. 663–690. With 5 coloured plates and 5 tables.

In a paper published in 1913 on the "Diagnosis of Latent Malaria," the authors suggested that the terms relapse and recrudescence should

be differentiated from each other. Relapse was defined as a biological phenomenon involving the re-formation of asexual forms from the and persistent gametes; whilst they regarded recrudescence as an resistant exacerbation of the asexual cycle.

As material from human sources was not available to afford proof of these views, experiments had to be carried out and investigations made regarding malarial infections in animals.

After having examined a number of monkeys for malarial infection without success and having found that sparrows infected with Proteosoma did not live in captivity for more than two or three days, the authors fell back upon *Halteridium columbae*, which was the only parasite analogous to *Plasmodium malariae* available.

From their studies in connection with the life-cycle of the parasite in the pigeon the authors have come to the conclusion that the description given by BEAUREPAIRE-ARAGAO is misleading, and that that given by NEGRI is correct for the mature schizont stage only. The figures given in DOFLEIN after LABBÉ are inaccurate, and in their opinion, represent the life-cycle of the Proteosoma parasite.

The details of the asexual cycle are said to be very puzzling, but the authors believe that their description of it is accurate in its essential details.

The asexual cycle.—The merozoites are small, ovoid or irregular bodies measuring about 2 microns in diameter, and consist of a small granule of chromatin with fairly darkly staining cytoplasm. They invade red corpuscles in the capillaries of the lungs and develop into trophozoites. These are oval in shape and measure from 3 to 5 microns in diameter. The chromatin is ovoid in shape and is situated near the narrower pole of the parasite. The cytoplasm stains faintly and contains very few pigment granules. They mature into schizonts.

The schizont rapidly increases in size and eventually obstructs the capillary in which it has been arrested, and the chromatin undergoes a process of fragmentation by binary fission. No pigment is observed in these stages, and in the centre of the schizont a large faintly stained mass of chromatin is seen.

The merozoites appear to escape by the bursting of the spore sac, and they carry on the asexual cycle by infecting fresh red corpuscles.

The asexual cycle appear to occur nowhere save in the lungs.

The merozoites give rise to gametes which require 6 to 10 days to come to maturity. These gametes are specially resistant forms. The author believe that only gametes are found in the peripheral blood because conditions there are not favourable for schizont formation, and that therefore young merozoites which are carried into the general circulation become gametes.

The macrogametocyte.—In this stage the parasite is elongated and it grows until it reaches the typical shape. The nucleus is centrally placed and stains a bright crimson colour. There are small light brown pigment granules scattered irregularly through the parasite.

The microgametocyte.—The young parasite is oval in shape, with a diffuse irregular orange staining chromatin mass. The cytoplasm is hyaline and stains of a faint purple colour. There may be one or two pigment granules, which are large. As the parasite increases in size the pigment granules become more numerous until 4 to 10 may be seen at each end of the parasite.

In opposition to the views expressed by CRAGG* in 1907, the authors state that (1) they have never seen intracorpuscular conjugation of trophozoites in the Halteridium; (2) The resting or "zygote" stage described by him has never been observed, but they consider the adult gamete as the resting stage and resistant form of the parasite, which alone is able to withstand injurious influences; (3) They regard the gamete as the originator of relapses.

The multiplication of the parasite in the capillaries of the lungs leads to the production of macroscopic changes in the appearance of these organs at the post-mortem, and they believe that in this fact there possibly lies an explanation of the frequency of pneumonia among malarial cachectics.

The authors believe that multiple infection of a single corpuscle is a matter of chance, and that though cytoplasmic union may occur each parasite maintains its individuality.

. During the winter 1912-13, the authors spent several months trying to confirm the asexual cycle as described by BEAUREPAIRE-ARAGAO, but entirely failed to do so. The reason for this was that they were working with pigeons which had been kept for several months under good conditions, and which had only mature gametes in their blood.

Early in 1913, a fresh batch of birds was obtained and in some of these young gametes were found in the peripheral blood. The authors were then able to work out the asexual cycle in the lung as described in this paper.

This observation was so repeatedly confirmed that they are able to rely upon the discovery of young gametes in the blood as an index to the presence of mature schizonts in the lungs. Where only mature gametes are present in the blood for a number of days it is found that asexual development in the lung has ceased.

A number of experiments were carried out in which blood was withdrawn from infected birds shewing either immature or mature gametes in their blood, the number of parasites being estimated from day to day. If there was an increase in the number after the taking of the blood it was an indication that asexual multiplication in the lung had not ceased and vice versa, because when only mature gametes are present in the blood there cannot be any production of new generations in the lungs by schizogony.

Though they have not been able to ascertain any maximum period for which gametes may persist in the blood, they think that it may be for some months or perhaps longer. By virtue of their great resistance and prolonged life the gametes enable the parasite to live through the winter months and bridge the interval from one fly season to another.

The authors made a number of observations upon the sex differentiation of the gametes present, and in 6 out of 10 pigeons examined the counts seemed to shew a typical Mendelian distribution; the female being dominant and the male the recessive.

In this disease the evidence points to the spleen as being the seat of destruction of the gametes.

In endeavouring to obtain evidence that female gametes can and do give rise to asexual forms, which would explain the occurrence of relapses after long intervals when fresh infection can be excluded,

* CRAIG is obviously meant.

the authors have used Bass's method of cultivating the halteridium of the pigeon.

Examination of the blood of a very large number of pigeons convinced the authors that it is in very exceptional cases only (they found one instance) that trophozoites can be found in the peripheral blood; they therefore made cultures from blood in which only gametes could be found on a number of successive days.

The blood was drawn from the marginal vein of the wing into an equal quantity of ·85 per cent. salt solution containing 1·5 per cent. of sodium citrate. This mixture was placed in sterile tubes containing 50 per cent. dextrose (Merck) solution in the proportion of 5 cc. of the mixture to 1–10 cc. of dextrose. The tubes were incubated in an atmosphere of hydrogen at 40°-42°C. and examined every two hours. Preparations were made from the layer of corpuscles immediately below the serum.

It appeared to be important that the blood should be allowed to cool in the syringe for a few minutes "to give the necessary stimulus to the process."

Appearances were found which indicated that asexual parasites did actually develop from the female gametes, and that these gave rise to merozoites.

In one instance an attempt was made to cultivate young gametes found in the blood to adult ones, but without success. The young gametes did not, however, assume a trophozoite-like form, thus shewing that the trophozoite-like forms which were observed in other cultures did not develop from young gametes.

The trophozoite forms appearing in the cultures were not present in the blood when taken from the bird, because control preparations shewed none, and, further, because ten out of twenty-one cultures shewed trophozoite forms, while trophozoites were only once found in the blood out of more than 200 pigeons examined.

The male gametes could not have taken part in the process as the conditions necessary for the formation of microgametes were not present and all the male parasites were observed to degenerate very rapidly in cultures and to be ingested by phagocytes.

The authors consider the change of temperature and environment form the stimulus to the female parasites to become parasites acting as trophozoites, and in this they find the explanation of the relapses occurring in malaria in the human subject, as such relapses always occur when circumstances become unfavourable to the host, and when the blood environment of the parasite (as for example when vaccines are introduced) is altered.

(344) Leger (A.) & Leger (M.). **Sur un Plasmodium de la Roussette du Haut-Sénégal et Niger.** [A Plasmodium of the "Flying-Fox" of the Upper Senegal and the Niger.]—*Compt. Rend. Soc. Biol.* 1914. July 24. Vol. 77. No. 26. pp. 399–401.

The authors have found parasites which they have identified with the parasite, *Pteropus gouldi*, described by Breinl in the Report of the Australian Institute of Tropical Medicine, 1911, as occurring in the Australian "flying-fox."

(345) Mackie (F. P.). **Note on the Parasite of Bat Malaria.**—*Indian Jl. Med. Research.* 1914. July. Vol. 2. No. 1. pp. 375–376. With 1 coloured plate.

The parasite described in this note was found in the blood of a flying fox (*Pteropus edwardsii*). The animal was kept under observation for several weeks without showing any symptoms of illness. The parasite closely resembled the malarial parasite of the African monkeys. The majority of those present in the blood were in the adult stage, and gametocytes were numerous. The addition of saline solution to the blood enabled the process of flagellation to be observed. The microgametes resembled those of the parasite of human malaria.

The infected animal was kept in a cage with others, but the infection did not spread.

In view of the fact that since the infected animal was found 56 others have been examined without success, it would appear that the parasite is somewhat rare. The name, *Plasmodium pteropi* (n. sp.), is suggested for the parasite.

HELMINTHS.

(346) Boynton (W. H.). **Kidney-Worm Infestation of Swine in the Philippine Islands with Special Reference to the Pathological Changes.**—*Philippine Jl. Science.* Sec. B. Trop. Med. 1914. June. Vol. 9. No. 3. pp. 269–289. With 3 plates.

The condition described in this paper is far more commonly met with in sub-tropical and tropical climates than in colder countries. It has been observed in the United States, especially in the southern part, South America, Australia, Asia, and in the Philippine Islands.

The cause of the condition is *Stephanurus dentatus*, also known as *Sclerostoma pinguicola*.

Examination of 2,000 pigs slaughtered at Manila showed that nearly 50 per cent. of the animals were affected.

The symptoms shown are: stiffness, especially of the hind limbs, the lameness and loss of power increasing progressively. There may be almost complete loss of power in the hind limbs a few days before death. The appetite is generally maintained until a few days before death.

The parasite has been encountered in a number of different situations. It occurs free upon, and embedded in, the fat surrounding the kidneys, and also within the kidney itself. It occurs free in the peritoneal cavity, in the liver, portal and mesenteric glands, and especially in great numbers in the connective tissue along the back. It has been encountered in the pleural cavity and embedded in the pleura.

It is said that in infested carcases putrefaction occurs very rapidly.

A detailed description is given of the changes produced in the organs resulting from invasion by the worm.

(347) LANE (C.). **Bursate Nematodes from the Indian Elephant.**—*Indian Jl. Med. Research.* 1914. July. Vol. 2. No. 1. pp. 380–398. With 12 plates.

The author of this paper has had the opportunity of examining material from six elephants and has found eight species of bursate worms, of which six are new and fall into six genera.

As full a description as possible is given of each, and the paper is illustrated with numerous plates.

FILTERABLE VIRUSES.

(RINDERPEST AND INFECTIOUS ANAEMIA OF HORSES).

(348) BOYNTON (W. H.). **A Preliminary Report of Experiments on the Cultivation of the Virus of Rinderpest in Vitro.**—*Philippine Jl. Science.* Sec. B. Trop. Med. 1914. Feb. Vol. 9. No. 1. pp. 39–44.

In preliminary experiments it was found that sterile blood drawn from infected animals maintained its virulence better under anaerobic than under aerobic conditions, and also that the virulence was maintained for a longer time when the blood was withdrawn within a day or two of the first rise of temperature than at later stages.

Experiments showed that neither red corpuscles, white corpuscles, nor blood platelets transmitted the disease, nor did they confer any immunity.

Investigations were then carried out to ascertain the smallest quantity of blood that would set up the disease. By the use of very fine calibrated pipettes it was found that 1/2970 cc. of virulent blood was capable of setting up the disease, but that 1/9060 failed. These data were useful in the cultivation experiments for comparison with the dilution of the original blood brought about by successive transfers in culture media.

In a number of media that were first tried it was found that the virus died in from 36 to 68 hours, but positive results were subsequently obtained with a modified form of the medium described by NENCKI, SIEBER, and WIJNIKEWITCH, and with the medium used by BASS and JOHNS.

Nencki's medium, which consisted of 900 cc. of water containing 100 grammes of peptone and 20 grammes of sodium chloride, yielded negative results.

This medium was modified by the addition of $33\frac{1}{3}$ per cent. solution of glucose to the peptone water in the proportion of 0·1 cc. to 10 cc. Immediately before use normal sterile defibrinated blood was added to the tubes in the proportion of 1 cc. to each tube of 10 cc. These tubes were then inoculated with 0·5 or 1 cc. of virulent blood, anaerobic conditions being produced by putting a layer of sterile paraffin oil on the surface of the medium. The tubes were incubated at 40° C. Subinoculations were made every three or four days, using sterile pipettes and transferring 0·3 to 0·5 cc.

The medium giving the best results was a modification of that used by BASS and JOHNS for the cultivation of malarial parasites. Glucose solution was added to sterile defibrinated blood taken from a susceptible animal in the proportion of 0·1 cc. to 10 cc. This medium was inoculated and then covered with sterile paraffin and incubated at 40° C.

The sixth transfer covering a period of 21 days in the blood glucose medium was found to be virulent in one instance, and in another the sixth transfer at 19 days was found to be virulent. In the salt peptone mixture inoculation succeeded with the fourth transfer at 12 and 13 days.

To ascertain whether the successful inoculations were due to original virus, calculations were made to ascertain the dilution, and in one case the virulent dose of the sixth transfer was calculated to contain 1/2,832,921 cc. of original virus. This suggests that there was an actual multiplication.

Nothing could be discovered regarding the morphology of the virus.

One animal which recovered from the disease induced by a fourth transfer is being hyperimmunised with cultures with the object of testing its serum. Experiments are also being carried out to test the possibility of devising a combined method of immunising with cultures and serum.

(349) BOYNTON (W. H.). **Experiments on the Cultivation of Rinderpest Virus as described by Baldrey.**—*The Philippine Jl. Science.* Sec. B. Trop. Med. 1914. June. Vol. 9. No. 3. pp. 259–268.

BALDREY'S paper, which was published in the *Journal of Tropica Veterinary Science*, Vol. 6, No. 1, 1911, contained an account of some experiments in which rinderpest blood was mixed with broth and incubated for 24 hours at body temperature, the liquid thus obtained being used for the hyperimmunisation of animals.

BALDREY arrived at the conclusion that an active toxin was produced in the broth, and that this toxin killed off the virus. It was found that it was best to inject the material intravenously, as if given subcutaneously it produced very severe inflammatory lesions.

The author of the present paper has repeated BALDREY'S experiments, and his conclusions, excluding references to particular experiments, are as follows:—

"1. In the case of two animals which died in less time than the incubation period of rinderpest, after injection of the Martin's broth culture, the autopsy findings of the tissues indicated death from a bacterial infection and not from rinderpest. All evidence points to the conclusion that the Martin's broth employed in these two cases was contaminated by bacteria prior to injection in the animals. The results are attributed to poor aseptic technique, and greater care in subsequent inoculations, where no such toxaemias were induced in the injected animals, support the conclusion.

"The symptoms, lesions, and other circumstances stated by BALDREY resemble the results obtained in the two animals in question, and there is justification for belief that his results were due to the same cause.

"2. In all the other animals injected with mixtures of blood and culture medium after incubation, no immediate ill effect followed in either susceptible or immune animals.

"3. With the exception of the animals noted in these experiments, all those injected with the so-called twenty-four- and forty-eight-hour cultures

of rinderpest in neutral or alkaline Martin's broth contracted rinderpest after the usual incubation period and died. These observations do not support BALDREY'S belief that there occurs a rapid formation of rinderpest toxin in the broth during the twenty-four hours with the resulting death of the virus. The experiments have included tests of Martin's broth after incubation as long as seventy-two hours.

"Rinderpest virus does die in Martin's broth culture after incubation for seventy-two hours, but there is no evidence that rinderpest toxin was formed, much less that rinderpest toxin caused the death of the virus.

"4. The experiments reveal the fact that rinderpest virus will survive in neutral or alkaline Martin's broth at 37°C. for at least forty-eight hours, but not for seventy-two hours. Two cases were tested at twenty-four hours, 2 at forty-eight hours, and 3 at seventy-two hours.

"5. Rinderpest virus kept in acid Martin's broth or in 5 per cent. potassium citrate solution did not survive after forty-eight hours at 37°C."

(350) BOYNTON (W. H.). **An Atypical Case of Rinderpest in a Carabao.**—*Philippine Jl. Science.* Sec. B. Trop. Med. 1914. Feb. Vol. 9. No. 1. pp. 45–47. With 1 chart.

The facts regarding the peculiar case of rinderpest described in this article are summarised in the conclusions and are as follows :—

"1. From the facts of this case, the evidence is conclusive that an animal may experience a fatal attack of rinderpest without the occurrence of a rise in temperature.

"2. The blood of the carabao was shown to be infected within forty-eight hours after it was originally injected with virulent blood.

"3. It was shown that the blood was virulent on the eleventh day when injected into a susceptible animal, yet exposure to the same animal from which blood was drawn did not cause rinderpest in the exposed susceptible animal.

"4. With regard to the three animals which failed to contract rinderpest by exposure, the question is raised as to whether rinderpest spreads by contact readily in the later stages of the disease or whether the disease must necessarily be accompanied by a rise of temperature before it can spread by contact."

(351) WARD (A. R), WOOD (W. F.) & BOYNTON (W. H.). **Experiments upon the Transmission of Rinderpest.**—*Philippine Jl. Science.* Sec. B. Trop. Med. 1914. Feb. Vol. 9. No. 1. pp. 49–78. With 2 plates and 6 charts.

The experiments described in this paper were designed to supply answers to the questions as to the period for which the virus can retain its vitality outside the body under varying natural conditions, and also the period during the course of the disease in which the virus is disseminated by sick animals.

The following conclusions were arrived at :—

"1. Rinderpest virus was not shown to have survived beyond twenty-four hours in corrals bare of vegetation but containing water. The conditions under which the tests were made included all seasons of the year, with accompanying variation in sunlight, rain, and condition of the soil. The amount of shade varied widely.

"2. Animals in such corrals became infected with rinderpest within half an hour, twelve hours, and seventeen and one-half, respectively, after the removal of the sick.

"3. Animals infected with rinderpest were shown to be capable of transmitting the disease to susceptible animals by close contact only during the febrile period of the disease, and most certainly during the period when the temperature was declining. The disease was not contracted

by susceptible animals when exposed to sick animals during the convalescent stage when the temperature was nearly normal.

"4. Blood of animals infected with rinderpest was shown in two cases to be infected during the height of the febrile period.

"5. The virus in urine, diluted with water and sprinkled on grass, was demonstrated to survive for thirty-six hours in some instances, but not always, and not for a longer period of time.

"6. Faeces mixed with water and sprinkled on grass infected an animal twenty-four hours later.

"7. Faeces and urine diluted with water and kept in a vessel in the shade remained infective for susceptible animals for thirty-six hours, but no longer.

"8. No evidence was secured to show that recovered cases transmit the disease.

"9. The foregoing facts indicate that the virus of rinderpest perishes soon after being discharged by the infected animal.

"10. Nothing in the foregoing experiments indicates that rinderpest virus is harboured for long periods upon the soil of contaminated areas."

(352) **Report on the Results obtained by the Special Committee for the Investigation of Infectious Anaemia among Horses.**—The Horse Administration Bureau. Tokyo. 1914. pp. 1–59. With 9 plates, 3 charts and 1 map.

The Special Committee, whose report this is, was appointed by the Japanese Government for the investigation of pernicious anaemia in horses in 1909. The personnel of the committee included representatives of the Horse Administration Bureau, the Imperial University at Tokyo, the Department of Agriculture, and the Army Veterinary Service.

Although exact information was not obtainable, it appears to be probably that infectious anaemia among horses was unknown in Japan prior to 1895.

The disease seems to be confined to Hokkaido, the northernmost island, and the north eastern part of Hondo, the main island. The southern portion of Japan is not invaded, because this portion of the country contains no pasture land, and it is among horses at pasture that the disease spreads.

Exact information is also lacking regarding the annual losses from the disease. In the first year of any outbreak the number of cases is small. This is followed by a progressive increase during the following years until the disease may be so severe that the majority of the horses in the district die. The disease then slowly dies down again. Once infected a district remains infected for a number of years, cases having been recorded in some districts for more than ten consecutive years after the initial outbreak.

In 1907, it was recognised that a number of deaths among the horses belonging to the Remount Depôt, which had been attributed to other causes in previous years, were in reality due to infectious anaemia. The maximum losses occurred in 1910 when a total of 500 horses died or were killed. Since that date the number of cases has decreased as a result of the preventive measures put into force. Since the virus is generally introduced by two-year-olds which have every appearance of being healthy at the time of purchase, but which afterwards prove to be infected, complete success in stamping out the disease among the remount animals has not yet been achieved, and fairly heavy losses still occur.

It is while horses are at pasture that the majority contract the infection, animals very rarely becoming infected in stables. Unless death occurs soon after infection, the disease takes a chronic course. During the first few months after infection repeated febrile attacks occur, but in cases in which the animal survives these gradually decrease in severity and finally cease.

The virus was found to be capable of passing through a Berkefield filter, and all attempts to cultivate it outside the body failed. Experimental inoculations shewed that the virus is present in the blood and all the organs, and also in the urine and milk, but attempts to prove its presence in the faeces and sweat of infected animals failed.

That the virus is protozal rather than bacterial in nature is indicated by the fact that the addition of 2·5 to 5 per cent. of sodium taurocholate to infective blood rendered it incapable of setting up the disease by inoculation.

Heating the virus to 60°C. for an hour was found to destroy its virulence. Exposure to bright sunlight (August) for two hours was also found to effect its destruction. Blood exposed out of doors from December to April, during which time the temperature was often as low as −9°C., was proved to have retained its virulence.

Blood was drawn from 21 horses at periods varying from 3 to 22 months after recovery and injected into healthy animals. All became infected with the exception of two which had received blood from horses which had recovered for three and thirteen months respectively. That these two animals were actually infected, although their blood failed to set up the disease, was proved by the inoculation of healthy horses with materials obtained from various organs when the animals were killed. Further experiments shewed that the blood may retain its virulence for periods ranging up to four years, but that the degree of virulence gradually diminishes.

Experiments were carried out to test the relationship between affected parents and their offspring with regard to the disease.

Blood drawn from four foals, the produce of infected mares, was injected into four healthy horses; one became infected, in two the symptoms produced were doubtful, and in the remaining one the result was negative. The injection of 30 to 100 cc. of milk from infected mares proved successful in three instances, but the milk of infected animals failed to set up the disease by ingestion.

That the progeny of infected mares do not acquire the infection through the medium of the mare's milk was proved by the fact that the intravenous injection of blood from a foal before it had sucked its dam set up the disease.

From this it is concluded that the infection is transmitted hereditarily.

In the donkey the disease generally follows an acute course and death takes place suddenly.

Among species of animals other than the equine the pig is the most susceptible to the virus. In the young goat and sheep a slight febrile condition is produced, but calves, dogs, rabbits, guinea-pigs, and rats are insusceptible.

Details of three experiments with pigs are given.

In the first the subcutaneous inoculation of 10 cc. of virulent blood was followed on the 11th day by a rise of temperature to 40·2°C.

There was interference with locomotion, loss of appetite, acceleration of the heart, frequent shivering fits and, finally, unconsciousness and death.

The second pig inoculated died suddenly during a febrile attack on the fifth day.

The third pig was inoculated with 10 cc. of virulent blood, but failed to shew any evidence of infection. A month later 20 cc. of its blood set up infection in a horse, proving that the virus had been in the pig for that period.

As already mentioned, infection occurs almost entirely while horses are at pasture, very occasional cases occurring in the stable. Experiments were therefore carried out to investigate the following points: Infectivity of the excreta of infected animals; infection through the alimentary canal; infection in the stable; and infection at pasture.

Experiments shewed that the disease could be transmitted by inoculation with infected urine, but no evidence was obtained that the virus was contained in the faeces.

It was also found that the disease could be produced experimentally by the administration of large quantities of infected urine (100 to 200 cc.) by the mouth.

A number of experiments in which 11 healthy horses were stabled with infected animals proved that infection in the stable is of rather rare occurrence. These experiments were carried out during the winter when there were no flies.

Principal importance attaches to the spread of the disease at pasture and in this connection the following points were investigated: The persistence of the virus in pasture during the winter; the manner in which infection occurs when healthy and diseased animals are kept together at pasture; and the relationship of blood-sucking insects to the disease.

In connection with the first of these points experiments shewed that it was quite safe to put healthy horses on to a pasture where numerous cases had occurred during the previous years, but that if healthy horses are pastured with diseased animals or animals that are carriers of the virus there is a very great risk that the healthy animals will become infected. In fact, in the experiments quoted the healthy horses contracted the disease in every instance. The shortest period of incubation was 16 days.

Although the virus is contained in the urine, it requires a large dose of this to produce infection experimentally and, further, the greater part of the urine passed soaks into the ground, that remaining on the top being disinfected by sunlight. Urine can therefore be excluded as the source of infection. This is also indicated by the fact that in stables, where the quantity of excreta is greater than in the open, the disease is far less commonly encountered.

To test the question as to whether blood-sucking insects play any part in the transmission of the disease a number of experiments were carried out.

In two experiments infected horses were kept with healthy ones in insect-proof enclosures for a period of some months. Two out of four healthy animals became infected from one diseased horse kept with them, and in the other experiment one out of two healthy horses

became infected when kept with four animals which were in a very advanced stage of the disease.

This proved that the disease could be transmitted without the agency of insects, provided the place in which the animals were kept was highly infected, but the rate of infection could not be compared with what occurs in the open pasture.

In the second series of experiments healthy and diseased horses were kept separate from each other in fenced areas separated from each other by a distance of about 15 yards. The animals could not mix with each other, but insects could pass freely between the batches.

Three experiments were carried out on these lines. In the first, nine infected horses were kept in one enclosure while four healthy animals were kept in the adjacent one. All the healthy animals contracted the infection.

In the second experiment five infected animals were placed in one enclosure. Two of these died shortly afterwards; one had a severe attack, and in the other two the symptoms were not very marked. Two healthy horses were kept in another enclosure during the whole season without becoming infected, but three out of four healthy animals placed in another enclosure, two at a time, became infected.

The second experiment was repeated the following year with the exception that nine infected animals were used instead of five. In this instance all the healthy animals contracted the disease.

From these experiments the conclusion is drawn that the disease can be transmitted without actual mixing of the diseased and healthy animals, and under conditions which exclude the intervention of insects other than those capable of flying.

Experiments with flies, ticks, etc.

Ticks.—Experiments showed that ticks taken from diseased horses and placed upon healthy horses never transmitted the infection.

Bot-flies.—These are not blood-sucking insects, but to exclude the possibility of the virus being taken into the stomach with the larvae, a number of the latter were taken from the stomach of a diseased horse, crushed in a mortar, and injected into healthy animals. In no instance was the disease transmitted. Further, it is stated that during one year bot-flies were exterminated from all the horses before pasturing, and it was noticed that this had no effect upon the prevalence of the disease.

Mosquitoes can be excluded, as they are found in the stables only, and not on the pastures.

Experiments showed that although sand flies are energetic blood-suckers they are not capable of transmitting the infection.

Negative results were also obtained in experiments in which *Stomoxys calcitrans* was used.

Tabanidae.—These flies are most abundant on the pastures, and energetically attack horses, and particular mention is made of the fact that the spread of the disease coincides with the appearance of this insect. It was found that the flies could not be kept alive for any length of time after they had been caught, and also that they could not be got to suck blood from the animals under experimental conditions.

Although direct proof is not forthcoming, the Committee feel compelled to conclude that the disease is spread through the agency of these flies.

Several species of horse-fly occur on the pastures where the experiments were carried out. The principal ones are the following:—*Chrysops japonicus, Haematopota tristis, Tabanus chrysurus, Tabanus trigonus, Tabanus trigeminus, Tabanus luridus, Tabanus rufidens,* etc.

The lesions present in cases of the disease vary in degree according as the case is acute or chronic, but not in character. They are typically those of anaemia, and in chronic cases there is marked emaciation. The visible mucous membranes are pale, and frequently ecchymosed. The subcutaneous and intramuscular connective tissue contains a gelatinous or blood-stained exudate. The muscles are soft, and pale in colour. The alimentary tract shows no lesions save for catarrh in some cases.

In acute cases, or after several attacks, the spleen may be two or three times the normal size. The capsule may show petechiae, and the pulp presents an appearance resembling that seen in cases of anthrax. In chronic cases the spleen is enlarged and firmer than normal. The capsule of the liver shows white patches of varying size and shape, these being particularly numerous between the lobules.

In acute cases the kidneys are enlarged and softened, and covered with small petechiae.

The lungs do not show any changes peculiar to the disease save oedema, but in some cases there is broncho-pneumonia.

The blood is very watery and greatly reduced in amount. The lymphatic glands, both of the carcase and of the viscera, are enlarged and oedematous, and of a reddish brown colour.

Histological examination reveals changes corresponding to the lesions: Anaemia, extravasations, degenerations of the parenchymatous organs, and the presence of haemoglobin crystals. Special mention is made of an infiltration of small round cells into the parenchymatous organs.

The prominent symptoms of the disease are the irregular occurrence of fever and progressive anaemia. In the majority of cases the fever recurs several times in a month, but sometimes periods of a few weeks up to six months elapse between consecutive febrile attacks.

At the onset of the disease the temperature generally rises suddenly to 39° or 40° C., and after remaining at that level for a day or two suddenly falls again. In chronic cases the temperature remains for some time at a subfebrile level.

During the febrile attacks the pulse rate may be as high as 90 per minute, but it maintains a moderate strength. In the last stages the pulse may be 120 and very weak. Very slight exertion causes an acceleration of the pulse and an increase in the force of the heart beats. Pulsation of the jugulars, which may extend half way up the neck, is frequently seen.

There are little or no disturbances in connection with the lungs, save that exertion causes a distinct acceleration of the respirations, and towards the end froth may be passed through the nostrils owing to oedema of the lungs.

During the febrile attacks there is always albumen in the urine, the

maximum being 10·5 per cent. During remissions and in chronic cases no albumen is found.

In severe or chronic cases the blood clots very slowly, in some cases several days elapsing before the process occurs.

Very few changes are observed in the morphology of the red blood corpuscles. There is a reduction in the number of white corpuscles, but this is not proportional to the decrease observed in the red corpuscles.

All attempts to discover a means of diagnosis apart from clinical methods and the experimental inoculation of susceptible animals failed. A similar result attended all efforts to find a drug which would influence the course of the disease.

Immunisation.—Seven recovered horses were treated with increasing doses of virulent serum and virulent blood in doses of from 2 cc. to 4,000 cc. at intervals of three to six days. Three of these animals survived the treatment, two died of the disease during the course of the immunisation, and in the remaining two cases treatment had to be stopped owing to the development of swellings at the seat of inoculation.

The serum from these immunised horses was found to be of no value as a protective agent, but actually a source of danger, as it was proved to contain the virus of the disease, and to be capable of setting up infection in healthy horses.

It was further found that if the material used as antigen contains blood corpuscles mixed with it a powerful isolysin is produced. Haemolysis occurred not only in infected animals which were treated with the serum, but also in a healthy horse, the count falling from 8,000,000 to 2,700,000 quite suddenly. Virulent serum heated to 60° C. for an hour was proved to be without protective properties.

A number of experiments were carried out with goats, pigs, and calves with the object of ascertaining whether any attenuation could be produced by passing the virus through these animals. No evidence of attenuation could be obtained. In the case of the goat and the calf the virulence appeared to be destroyed.

The addition of 2 per cent. carbolic acid to serum was not sufficient to destroy the virus in less than an hour. Chloroform and toluol failed to destroy it even after remaining in contact with it for 20 hours.

The Report contains a summary of the laws and regulations laid down for the control of the disease.

MISCELLANEOUS.

(353) Laveran (A.) & Franchini (G.). **Infections de Mammifères par des Flagellés d'Invertébrés.** [The Infection of Mammals with the Flagellates of Invertebrates.]—*Bull. Soc. Path. Exot.* 1914. July. Vol. 7. No. 7. pp. 605–612. With 4 text figures.

In previous papers (*Compt. Rend. Acad. Sci.*, 1913, September 1st and November 4th, and 1914, March 16th) the authors have published their observations regarding the infection of mice and white rats with

flagellates derived from the dog flea, the rat flea, *Anopheles maculipennis*, and *Melophagus ovinus*.

Natural infections have been observed to take place in the mouse and rat, and it has been found possible to infect mice by causing them to ingest rat fleas containing flagellates.

Figures are given showing the principal forms in which *Herpetomonas ctenocephali* occurs, and it is said that the flagellates of the rat flea differ but little from those of the dog flea. Rounded or leishmania-forms and flagellate forms occur.

In the case of *Crithidia fasciculata*, the flagellate of *Anopheles maculipennis*, crithidial forms generally predominate but, mixed with these, forms possessing all the characters of the genus Herpetomonas are to be found.

The crithidial form also predominates among the flagellates of the intestine of *Melophagus ovinus*.

With whichever of these flagellates the mice or rats are infected the course of the infection is practically the same.

At first the blood is found to contain small oval or rounded intracellular bodies measuring about 2 microns in diameter. The smallest bodies possess only nucleus, but in the larger ones a nucleus and centrosome can be made out. A certain number of these parasites may be observed to be in a state of division.

The "Anaplasma forms" which may be found in the blood of young animals do not appear to be of a parasitic nature.

At a later stage of infection occasional extra-cellular forms may be found. These are fusiform and non-flagellated. They are about five microns in length and possess a nucleus and centrosome.

In a number of cases infection terminates fatally, although parasites are never present in large numbers in these animals. In preparations made from the liver, spleen, and bone marrow free leishmania-like forms predominate. In some preparations from the liver the authors have occasionally observed oval or spherical forms measuring about five microns. In these a nucleus and centrosome can be distinguished, the latter forming the starting point of a flagellum measuring about 12 microns.

Further fresh points are brought forward in the present paper.

Brief details are given of experiments which indicate that it is easy to infect young white mice by allowing them to ingest the faces of rat fleas which contain *Herpetomonas pattoni*.

Endocellular parasites appear in the blood about the third or fourth day, and flagellated parasites a few days later.

Two experiments to infect dogs with the flagellates of the dog flea and the rat flea failed.

A monkey inoculated intraperitoneally with emulsion of the spleen of a mouse infected from rat fleas showed parasites in its blood after about 5 weeks.

In a mouse inoculated with *Crithidia fasciculata* there developed a skin lesion resembling oriental boil, and preparations made from the deep parts of the lesion revealed the presence of leishmania-like bodies. The centrosome of these bodies was rarely bacilliform.

Attempts to cultivate the organisms gave very doubtful results.

The authors conclude that the evidence obtained supports the view that the leishmaniae and the trypanosomes of the vertebrates may have their origin in the flagellates of the invertebrates.

(354) Holmes (J. D. E.). **Bursati.**—*Memoirs Dept. Agric. in India.* Veterinary Series. 1914. Sept. Vol. 2. No. 5. pp. 119–153. With 5 plates comprising 11 figures.

This memoir is divided into five parts dealing respectively with the following points: History of Bursati; the Identity of Bursati with Leeches, Summer sore, Granular dermatitis, and other skin affections; Clinical characteristics of Bursati; Histology and Etiology of Bursati; and the Treatment of the condition.

There is also included a list of references and a number of plates, some of which are coloured.

The first part gives some dozen brief references to the opinions expressed by various authors at different times during the last century as to the nature of the disease.

Part II. summarises briefly the views of a number of observers regarding the identity or otherwise of bursati with a number of skin diseases occurring in other parts of the world than India.

Part III. The most accurate description of the clinical features of the disease appears to be that published by Smith (1874 and 1884). Smith's account is given at some length and the principal points are the following: The majority of cases occur in the rainy season, but many cases are encountered during the hot weather. The parts generally involved are the angles of the mouth, prepuce, glans penis, fetlocks, and pasterns. The morbid processes start in the subcutaneous tissue. The most distinctive feature of the disease is that it becomes indolent in character, the products of the disease undergoing a peculiar elaboration and remaining as irritants. These products are the so-called "Kunkur stones."

The progress of the formation of a lesion is most easily followed at a place where the skin is thin. There is first of all swelling, and after a few days it is possible to detect that at one point the swelling is becoming harder than elsewhere. This increases in size until it is about the size of a shilling. The skin over the hardened area then becomes thickened and adherent to the tissue beneath. This occurs after about a fortnight. The cuticle is removed either by friction or otherwise and an open sore results. The acute inflammation suddenly disappears and the diseased process becomes an indolent one. The edges of the sore are raised above the surface, are fairly regular and sometimes callous. The ulcerating surface is concave, and situated on and embedded in it are yellowish spherical bodies, which can be pressed out leaving depressions behind. From these depressions a clear liquid exudes. These are the "kunkur bodies."

Although it is common to regard bursati as the result of wound infection, Holmes states that that is not the most common way in which a bursati lesion is started. He is in agreement with Smith that the lesion is in the first place one of the subcutaneous tissue, and that it is only in the second stage that the skin becomes involved. By the time the skin is lost the underlying tissue has become hard and the surface covered with granulations. In situations where the lesion is not subjected to friction there is little tendency for it to spread, or for the granulations to become very prominent. The surface is dry and sometimes covered with a scab. There may be a

slight serous discharge but, except in old lesions where secondary invasion by pyogenic bacteria has taken place, there is no pus.

Bursati lesions involving mucous membranes are found on the inner canthus of the eye, the angle of the mouth, and the penis. Irregular forms of the disease sometimes occur in which the epidermis is diffusely eroded without any deep-seated lesions being formed.

Holmes has never seen cases of the disease in which there were lesions of the internal organs such as have been described by BURKE, MEYRICK, LINGARD, and others, and he believes that these were cases of mistaken diagnosis.

From the description given it appears that the condition is quite distinct from "leeches," granular dermatitis, summer sores, and other similar skin affections. It is stated that a disease somewhat similar to bursati occurs in America; but there appears to be little doubt that there is some confusion regarding the correct diagnosis of "leeches" and that more than one disease has been described under this name.

In the case of summer sore the calcareous nodules have a very different structure to the kunkurs found in bursati. In summer sore the nodules have an outer covering of fibrous tissue and a central cavity which has a calcified wall.

In bursati the outline of the lesions is not regular, and the kunkur is easily enucleated from the fibrous capsule. The inner mass is hard and fibrous, and is neither caseous nor to any extent calcareous. When pressed between two glass slides it behaves like a hard piece of rubber.

Part IV.—The primary process in the formation of a bursati sore is an area of subcutaneous inflamation due to some specific agent. This irritation, which occurs in a part which has previously had its vitality lowered by some traumatic cause, sets up necrosis of the skin, While this is going on the subcutaneous tissue becomes hard and infiltrated. When the necrotic skin is shed there is left a circular or oval sore with well defined edges. Granulations soon appear in the central part and cause this to become elevated above the surrounding tissues. If such a lesion is kept free from irritation of all kinds it shews little or no tendency to spread, but at the same time no tendency to heal. In debilitated animals, or in cases where the lesion is exposed to irritation, it shews a tendency to spread with the resulting formation of a diffuse tumour in the subcutaneous tissue. The granulations on the surface are very vascular, and pressure causes the expulsion of kunkurs as large as a pin's head or a grain of shot. There is no degeneration of the tissues or pus formation except after invasion with pyogenic bacteria. The deeper fibrous tissue has kunkurs embedded in it and merges gradually into the surrounding tissues.

Microscopical examination of the lesions shews that they are composed of fibrous tissue. In the younger ones there are large numbers of cells of different kinds, and particularly of eosinophiles. In older lesions the fibrous tissue is more dense and giant cells are found. Spaces are found in the sections from which kunkurs have dropped out. These spaces are surrounded by fibrous tissue and in some sections a few pigment-bearing cells may be found. There is no evidence of calification or other form of degeneration.

The author has been unable to find any evidence that the lesions are caused by filarial embryos, as stated by LINGARD. He has,

however, been able to find a fungoid organism which he believes is responsible for the condition.

The most satisfactory method of examination is as follows: Small blocks of the tumours or kunkurs are washed in alcohol, ether, and water, and then macerated in 40 per cent. caustic potash for 16 to 20 hours. A fragment of such material is taken and pressed between two slides. Isolated spore-like bodies and broken strands of mycelia can be found, but they cannot be satisfactorily differentiated from fragments of cells, granules of eosinophiles and tissue fibres. The most convincing pictures are obtained with material from young lesions. In these can be seen groups of spores which on careful examination are found to be double contoured. The organism is difficult to demonstrate by staining, as it does not take the stain well and is easily decolourised.

Cultivation on Sabouraud's agar most constantly yielded a slow-growing fungus. Growth first became evident on the fifth day in the form of a small dry-looking colony. This gradually spread until the whole surface was covered with a white chalk-like substance which was easily removed, leaving a dry opaque skin adhering to the agar. Cultures of this nature were obtained both from bursati tissue and blood from infected horses.

BUTLER found that the fungus most closely resembled *Sporotricum minutissimum*.

All attempts to infect horses, guinea-pigs, and rabbits failed.

Part V.—The treatment of the condition.—For local treatment the lesions must be completely excised, the wounds being treated as ordinary wounds.

In the experimental treatment of bursati some success has been obtained with arsenic either in conjunction with atoxyl or alone, the treatment being carried out after the manner employed for surra. Under this treatment some cases shewed a rapid improvement, while others did not appear to be benefited.

Some such cases shewed rapid recovery when subjected to treatment with biniodide of mercury and potassium iodide, the doses being 5 grains increasing to 20 of the former and 1 to 3 drachms of the latter. Three courses were given of ten to 12 days each with intervals of one week.

HAZELTON recommends that after the diseased tissues have been surgically removed the cavity should be kept constantly dressed with crystalline red oxide of mercury.

Conclusions:

"1. A considerable amount of confusion exists in literature regarding the identity of Bursati in India with 'Leeches' in America, 'Swamp Cancer' in Australia and other affections described as 'Summer Sore,' 'Granular Dermatitis' and 'Parasitic Fibromata,' etc.

"2. The latter affections such as 'Summer Sore,' 'Granular Dermatitis' and 'Parasitic Fibromata,' etc., are held, on the observations of several authors, to be connected with the presence of nematode embryos.

"3. Bursati varies in most clinical aspects from the affections described as 'Summer Sore,' 'Granular Dermatitis' and 'Parasitic Fibromata,' etc.

"4. There is not sufficient evidence to prove that nematode embryos are present in Bursati lesions or that Bursati sores or tumours are caused by such embryos.

"5. There is a similarity in several clinical aspects between 'Leeches' and Bursati.

"The presence of a fungus in 'Leeches' has been recorded by several observers and described as the causal agent. The spores and mycelia of a somewhat similar fungus are frequently found in the kunkur and tumours of Bursati.

"6. The presence of spores and mycelia in the Bursati tumours, and the fact that the cultures of a fungus of the genus Sporotricum have repeatedly been obtained from the Bursati tumours and kunkurs and also direct from the blood of horses infected with Bursati, affords some evidence that the disease is a mycosis somewhat resembling the Sporotricosis of the horse and mule described by CAROUGEAU in Madagascar."

The paper includes 5 plates comprising 11 figures, shewing the appearance of lesions, spores, and mycelia, and cultures.

(355) HOLMES (J. D. E.) **Some Experiments on the Immunising Effect of the Simultaneous Injection of an Anthrax Attenuated Virus and an Anthrax Anti-serum.**—*Memoirs Dept. Agric. India.* Veterinary Series. 1914. Sept. Vol. 2. No. 7. pp. 199–206.

The experiments recorded in this paper were carried out in order to obtain information regarding the risks attaching to the use of attenuated cultures for the vaccination of animals against anthrax.

Vaccines were prepared by the various known methods, and were tested upon rabbits, guinea-pigs, and sheep, but with none of them was it possible to exclude mortality due to the inoculation. This was especially the case with the sheep. In some cases death from anthrax occurred as late as 20 days after inoculation, the virus apparently regaining its full virulence in the bodies of the animals.

Indian cattle, although susceptible to natural infection by ingestion, are very resistant to experimental inoculation, and therefore cannot be used for testing sera and vaccines.

In the first experiment a number of guinea-pigs, rabbits, sheep, and ponies were inoculated with broth cultures which had been incubated at 37° C. for 48 hours and then heated at 80° C. for one hour. This vaccine was administered in doses of 0·25 to 1 cc. to guinea-pigs and rabbits, 1 to 5 cc. to sheep, and 5 cc. to ponies. Four rabbits were used, and one which received the smaller dose died. Similarly, one guinea-pig out of three used died. Four sheep out of 12 succumbed, but all the three ponies used survived. The unheated culture proved fatal to three sheep in doses of 0·025 cc.

In the second series of experiments a number of sheep were inoculated with 5 cc. of this vaccine and simultaneously with doses of 5 to 25 cc. of a protective serum, which was of such strength that 20 cc. was protective for sheep against inoculation with virulent anthrax. Two of the sheep died in about a week. The surviving five animals were inoculated after eight weeks with 0·025 cc. of virulent culture. All survived.

In the second series of experiments of the same nature 12 sheep received a full dose of protective serum with 1 cc. of vaccine. Two of these died in nine days. One died when the immunity was tested eight weeks later, and one more died when again tested after a further interval of six weeks.

A third series of eight sheep were subjected to a similar experiment, save that the dose of vaccine was 5 cc. All survived the vaccination, but three died within five days when their immunity was tested after an interval of eight weeks.

Tests on ponies.—Eight ponies, ranging from 300 to 400 lb. body weight, were given from 15 to 50 cc. of serum and 5 cc. of vaccine. None died. After eight weeks two succumbed to inoculation with 0·025 cc. of virulent anthrax culture within nine days. Two showed temperature reactions, but lived.

In this experiment one of the three virus control ponies survived.

Five donkeys survived vaccination with 25 cc. of serum and 5 cc. of vaccine, but one died in five days when their immunity was tested with 0·5 cc. of virus after an interval of eight weeks.

Five sheep were inoculated with 5 cc. of vaccine alone. On being tested with 0·025 cc. of virus after an interval of three weeks, all died within three days.

Conclusions :—

"1. The method of immunisation against anthrax by means of the simultaneous injection of serum with the inoculation of an attenuated culture is not free from risk. A varying number of inoculation mortalities follow.

"2. Sero-vaccination confers an active immunity. It does not, however, protect in all cases treated, and the results are variable.

"3. An inoculation of the vaccine alone did not confer any immunity.

"4. The use of an attenuated culture in combination with anti-serum does not appear to be a method in any way superior to the combination of a virulent culture with anti-serum. Following both methods a varying percentage of mortalities from Anthrax occurs, and although an active immunity is produced in the animals which do not succumb, this immunity does not extend to all cases, and a certain number of animals remain susceptible to Anthrax infection."

(356) Cummins (S. L.), Coppinger (C. J.) & Urquhart (A. L.). **Further Observations on the Presence of Antibodies for *Micrococcus melitensis* in the Milk of English Cows.**—*Jl. Royal Army Med. Corps.* 1914. July. Vol. 23. No. 1. pp. 36–41.

In the *Journal of the Royal Army Medical Corps* for January 1914, Kennedy drew attention to the fact that the sera of five out of 22 English cows examined contained agglutinins for the *Micrococcus melitensis*. The milk of three of these animals gave positive results in high dilutions, and the whey obtained from these milks yielded results exactly similar to those given by the milks. Filtration of the milk or whey appeared to diminish the agglutination. Bassett-Smith found one sample of milk out of a number examined which caused agglutination of the organism in dilutions up to 1 in 100. Attempts to cultivate the micrococcus from the milk failed in every case.

These results are of the greatest importance because, if further investigation shows that the reaction is not connected with infection with the organism, the value of the test in the detection of infected goats will have to be reconsidered. If there is found to be a connection between the reaction and infection of English cattle, the question of the infection of the milk will acquire great importance.

The agglutination of the micrococcus by cow's milk has already been observed in Gibraltar, this having been recorded by Horrocks in 1907. The infection of cows was also referred to in the Report of the Commission on Malta Fever, Part 1, in 1905.

The present paper records the results obtained during the examination of the milk of seven cows, two of which gave positive results.

One of these yielded milk which agglutinated the micrococcus in dilutions varying from 1 in 250 to 1 in 1,000 on different dates.

It was ascertained that a degree of acidity many times higher than that of milk does not cause agglutination of the organism, and that therefore acidity is not a factor in the production of the agglutination.

The milk, whey, and blood serum of the cow all agglutinated the organism to corresponding dilutions. The agglutinin were demonstrable in the milk when tested almost immediately after drawing.

It was also found that heating either the milk, whey or serum to 57° C. for 25 minutes did not destroy the agglutinin, but that the optimum concentration of the agglutinating substance was moved to higher dilutions by heating; thus paradoxal results were obtained. Such paradoxal results were not obtained with unheated milk. This effect was most marked with the blood-serum.

The milk failed to agglutinate *B. typhosus, coli, dysenteriae,* and *M. paramelitensis.* The whey did not lose its agglutinating power after being kept for three weeks.

In one experiment the passage of diluted whey (1 in 12) through a Doulton filter retarded, but did not eliminate, the agglutinin. The serum of an immune rabbit diluted to the same degree when passed through a filter lost none of its agglutinating power. The rabbit serum when diluted with the whey from the cow and filtered still agglutinated to the same degree, but when whey from a control cow was added complete removal of the agglutinins resulted. These observations are to be repeated, as filters appear to differ in their power of arresting agglutinins.

The milk, whey, and serum of this cow all contained thermo-stable opsonins for the organism. The opsonin was present in large amounts, and was demonstrable in high dilutions. In dilutions up to 1 in 40 the number of cocci per cell were too numerous to allow of accurate enumeration, while with the control serum the number per cell in dilutions from 1 in 5 to 1 in 320 was about 0·5.

Opsonin was still demonstrable after the whey had been kept for three weeks.

The authors have not been able to demonstrate the presence of deviating substances in the heated milk or whey.

In one experiment the heated serum was able completely to deviate two minimum haemolytic doses of complement up to a dilution of 1 in 16. The dilutions were not carried further. In a second experiment no deviation was obtained, but in this instance the serum had been kept diluted with saline for several days, and a positive rabbit serum also yielded negative results when treated in the same way.

The conclusion drawn is:—

The milk, whey, and blood serum of Cow 5 behaves towards *M. melitensis* in a manner comparable to the body-fluids of animals suffering from, or immunised against, this organism, due allowance being made for differences in concentration of "anti-substances" and degrees of immunity.

REPORTS.

(357) Hutchens (E.). **Annual Report of the Department of Agriculture. Uganda Protectorate. Year Ending March 31st 1914. Report of the Veterinary Department.** pp. 28–35. 1914. Kampala. The Uganda Printing and Publishing Company, Ltd.

Rinderpest.—During the year this disease has occurred for the first

time in a district west of the Nile, but up to the time of writing it had been confined to within 25 miles of the river and to the south of the Jinja-Kampala road.

Fifteen cases of the disease were treated by RICHARDSON with permanganate of potash on the lines suggested by WALKER [see this *Bulletin*, Vol. i. No. 2, p. 127.] The results were most hopeful and warrant an extended trial of the method in the treatment of individual cases.

Foot and mouth disease.—The disease is well-known in the Protectorate, but except in neglected cases in transport animals it is not responsible for serious losses.

Trypanosomiasis.—It has been proved by Miss ROBERTSON that *G. morsitans* is the transmitting agent of *T. pecorum* in Uganda. A number of hitherto unrecorded *morsitans* areas have been discovered, among which are Buruli, and two areas in Southern Ankole.

T. nanum was found in a donkey brought down from the Northern Province, and after three months' treatment (method not given), it appeared to have recovered. Trypanosomes of the *T. brucei* type were found in an Egyptian donkey and in a mule. Both animals died, the disease running a rather acute course.

Piroplasmosis in dogs has a wide distribution, but cases met with are seldom fatal if treated with trypanblue.

No cases of rabies have been found.

(358) BEVAN (Ll. E. W.). **Report of the Veterinary Bacteriologist, Southern Rhodesia for the Year 1913.** f'cap. 3 pp.

During the year under review little or no research work has been done, as Bevan was on vacation leave for eight months.

African Coast Fever.—Several outbreaks of this disease occurred. In some instances the circumstances were difficult of explanation. In more than one instance a single case cropped up, generally in a young animal. In other cases outbreaks occurred on old infected areas which according to the accepted theories should have long since been clean. One explanation offered is that an endemic form of the disease allied to the condition known as amakebe in Uganda exists in the Territory. Another is that recovered animals may act as reservoirs of infection.

Anaplasmosis has acquired an increased importance during the year. Not only has it been responsible for a number of deaths among imported animals, but it causes very severe losses in growth and development, which render it one of the greatest handicaps to stock raising. The resistance to the disease possessed by indigenous animals decreases when these are improved by crossing with imported animals, and as the breeding-up extends the disease will acquire more and more importance.

While redwater, the disease with which anaplasmosis is generally associated, is amenable to treatment with trypanblue, anaplasmosis has resisted all methods of treatment tried up to the present.

During the year, 94 animals imported from Great Britain were inoculated, and of these 27 died. In one instance 16 died out of a batch of 33. The deaths took place between the 35th and the 40th day after inoculation. The animal that was the source of the blood

for inoculation was a standard animal which had previously yielded a comparatively mild virus, but on examination its blood was found to contain spirochaetes in addition to anaplasms and piroplasms. It appears to be probable that this infection was acquired naturally through the medium of tick-infested bedding.

A number of experiments were carried out in connection with the immunisation of horses against horse sickness, and the results were considered to be very satisfactory when the animals were tested with very large doses of a certain virus. Unfortunately the animals had to be sold and all trace of many of them was lost, but there is no doubt that under careless management many of them died of the disease. The results, in those cases in which the animals could be followed, indicated that immunisation of an animal against one strain of the virus does not protect against all strains.

A heavy trypanosome infection occurred among trek oxen working in the " fly " area in the Hartley district, but good results were obtained with treatment (method not given). Treatment in pigs has not been successful.

A trypanosome identified by BRUCE as *T. caprae* has been found in the blood of a sheep from Mafungabusi and also in an ox in the Gatooma district.

RECENT LITERATURE.

[Continued from this *Bulletin* Vol. 2, No. 3, pp. 154–156.]

Anaplasmosis and Babesiasis.

(359) TIBADI (Ettore). Forme di Anaplasma nel Sangue di Diversi Animali in Sardegna. [Types of Anaplasma in the Blood of Various animals in Sardinia].—*Pathologica.* 1914. May 15. Vol 6. No. 133, pp. 261–264. With 1 text fig.

(360) VALLADARES (J. F.). Equine Biliary Fever in Madras.—*Parasitology.* 1914. May. Vol. 7. No. 1, pp. 88–94.

Rabies.

(361) NICOLLE (C.). La Rage en Tunisie pendant l'Année 1913. [Rabies in Tunis during the year 1913.]—*Arch. Inst. Past. Tunis,* 1914. Aug. 1. Vol. 9. No. 1, pp. 60–61.

Snake Bite.

(362) ACTON (H. W.) & KNOWLES (R.). Studies on the Treatment of Snake-Bite. Parts I, II and III.—*Indian Jl. Med. Research,* 1914. July. Vol. 2. No. 1, pp. 46–148. With 5 plates and 3 charts.

(363) BANNERMAN (W. B.). Report on the Treatment of Snake-Bite with Potassium Permanganate.—*Indian Jl. Med. Research,* 1914. July. Vol. 2. No. 1. pp. 149–182.

Spirochaetosis.

(364) CARPANO (Matteo). Ueber einige in papillomatösen Neubildungen bei Pferden aufgefundene Spirochäten. [Spirochaetes found in Papillomatous Tumours in Horses.]—*Centralbl. f. Bakt.* 1 Abt. Orig. 1914. July 25. Vol. 74. No. 7, pp. 584–591. With 1 plate and 18 text figs.

(365) WOLBACH (S. B.) & BINGER (C. A. L.). The Cultivation of a Free living Filterable Spirochaete (*Spirochaeta elusa:* New Species). A Preliminary Report.—*Jl. Med. Research.* 1914. Mar. Vol. 30. No. 1. pp. 9–22. With 3 plates comprising 18 figs.

(366) WOLBACH (S. B.) & BINGER (C. A. L.). Notes on a Filterable Spirochaete from Fresh Water. *Spirochaeta biflexa* (New Species).—*Jl. Med. Research.* 1914. Mar. Vol. 30. No. 1. pp. 23–25. With 1 plate comprising 15 figs.

Tick Paralysis.

(367) NUTTALL (G. H. F.). "Tick Paralysis" in Man and Animals. Further published Records, with Comments.—*Parasitology,* 1914. May. Vol. 7. No. 1, pp. 95–104.

Trypanosomiasis.

(368) LAFONT (A.) & DUPONT (V.). Sur les Résultats éloignés du Traitement de la Maladie du Sommeil par le Ludyl et le Galyl. [The Results of the Treatment of Sleeping Sickness by Ludyl and Galyl after considerable Intervals.]—*Bull. Soc. Path. Exot.* 1914. July. Vol. 7. No. 7, pp. 640–647.

Biting Flies.

(369) Cummings (B. F.). Descriptions of five new Species of Anoplura and Mallophaga.—*Bull. Entomol. Research*, 1914. Sept. Vol. 5. No. 2, pp. 155–177. With 8 text figs.

(370) Edwards (F. W.). New Culicidae from Borneo and Hong Kong.—*Bull. Entomol. Research.* 1914. Sept. Vol. 5. No. 2, pp. 125–128.

(371) Gough (L. H.). Preliminary Notes on Egyptian Mosquitos.—*Bull. Entomol. Research.* 1914. Sept. Vol. 5. No. 2, pp. 133–135.

(372) Mansion (G.). Les Phlébotomes européens. [The European Phlebotomi.]—*Bull. Soc. Path. Exot.*, 1914. July. Vol. 7. No. 7, pp. 584–590. With 7 text figs.

(373) Marzinowsky (E. I.). De l'Existence de *Stegomyia fasciata* (*St. Calopus*) en Russie. [The Occurrence of *Stegomyia fasciata* in Russia.]—*Bull. Soc. Path. Exot.*, 1914. July. Vol. 7. No. 7, pp. 590–593.

(374) Roper (R.). An Account of Some Anopheline Mosquitos found in British North Borneo, with Description of a New Species.—*Bull. Entomol. Research*, 1914. Sept. Vol. 5. No. 2, pp. 137–148. With 2 maps and 1 text-fig.

(375) Stanton (A. T.). The Anopheles of Malaya. Part II.—*Bull. Entomol. Research*, 1914. Sept. Vol. 5. No. 2, pp. 129–132. With 2 text-figs.

Helminths.

(376) Perroncito (E.). Nota sul Gastrodiscus. [Notes on Gastrodiscus].—*Giorn. R. Accad. Med. di Torino*, 1914. May–June. Vol. 77. No. 5–6, pp. 168–169.

(377) Perroncito (E.). Bronco-polmonite verminosa in Africa. [Verminous Broncho-Pneumonia in Africa].—*Giorn. R. Accad. Med. di Torino*, 1914. May–June. Vol. 77. No. 5–6, p. 170.

(378) Romanovitch (M.). Microfilaire des Chevaux atteints de Boutons Hémorragiques. [Microfilaria in Horses affected with Haemorrhagic Dermatitis].—*Compt. Rend. Soc. Biol*, 1914. July 24. Vol. 77. No. 26, pp. 390–391.

(379) Saunders (P. T.). Notes on some Parasites of Live Stock in the West Indies.—*West Indian Bull.*, 1914. Vol. 14. No. 2, pp. 132–138.

(380) Seurat (L.-G.). Sur un Nouveau Spiroptère du Chat Ganté. [A New Spiroptera of *Felis ocreata* Gmel.]—*Compt. Rend. Soc. Biol.*, 1914. July 17. Vol. 77. No. 25, pp. 344–347. With 5 text figs.

(381) Weiss (A.). Note sur les Microfilaires du Rat à Trompe. [The Microfilariae of the Elephant-Shrew.]—*Arch. Inst. Past. Tunis*, 1914. Aug 1. Vol. 9. No. 1, pp. 50–51.

Protozoa.

(382) Martini. (E). Ueber die Entwickelung von Malariaparasiten in Bassschen Nährboden [The Development of Malarial Parasites in Bass's medium]—*Centralbl f. Bakt.* I. Abt. Orig. 1914. June 13. Vol. 74. No. 3–4, pp. 250–254. With 2 coloured plates and 1 text fig.

(383) Coles (A. C.). Blood Parasites found in Mammals, Birds, and Fishes in England.—*Parasitology*, 1914. May. Vol. 7. No. 1, pp. 17–61. With 4 plates.

BRITISH MUSEUM 6 JAN 15

INDEX OF AUTHORS.

The numbers in heavy type indicate Abstracts, and those in light type References (Recent Literature).

A.

Acton (H. W.), **343**, 362.
Arnheim (G.), **169**.
Austen (E. E.), 213, 301.

B.

Balfour (A.), **285**.
Bannerman (W. B.), 363.
Bauche (J.), **30**, **185**, **186**, **187**, **278**.
Battaglia (M.), **19**.
Beck (M.), **144**.
Béguet (M.), **2**, **268**.
Behrend (K.), **174**.
Bernard (P. N.), **30**.
Bertarelli (E.), **73**.
Bevan (Ll. E. W.), **358**.
Binger (C. A. L.), 365, 366.
Blacklock (B.), **25**, **29**, **134**, **232**, **237**, **244**, **245**, **255**.
Blanchard (M.), **47**, **140**.
Bockŏer (E.), 206.
Bondeaud (A.), 92.
Boquet (A.), **3**.
Borrelini (A.), 202.
Botelho, jnr. (C.), 298.
Bouilliez (M.), **58**.
Bouquet (A.), **284**.
Bovone (E.), **23**.
Boynton (W. H.), **346**, **348**, **349**, **350**, **351**.
Braddon (W. L.), **74**.
Bronfenbrenner (J.), **266**.
Brown (W. H.), **241**, **242**.
Bruce (D.), **14**, **15**, **16**, **17**, **32**, **57**, **233**, **236**, **321**, **322**, **325**, **326**, **327**.
Bruce (Lady), **14**, **15**, **16**, **17**, **32**, **57**, **233**, **236**, **321**, **322**, **325**, **326**, **327**.
Brumpt (E.), 207.
Buck (J. M.), **31**.

C.

Cannata (S.), 97.
Carini (A.), **63**, **171**, 292, 298, 299.
Caronia (G.), 98, **256**.
Carpano (M.), **8**, **128**, **129**, 312, 313, **318**, 364.
Carpenter (G. D. H.), 114.
Chalmers (A. J.), 115, **323**.
Chambers (F.), **5**, **229**.
Ciuca (A.), **146**.
Ciuca (M.), **330**.
Clandon, 314.
Coles (A. C.), 383.
Combiesco (D.), **282**.
Cominotti (L.), 122.
Connal (A.), **267**.
Conor (A.), 220.
Conor (M.), **260**.
Coppinger (C. J.) **356**.
Couvy (L.), **49**.
Cross (H. E.), **329**.
Cruickshank (J. A.), **179**.
Cummings (B. F.), 369.
Cummins (S. L.), **356**.
Cunliffe (N.), 216, 217, 218.

D.

Danysz (J.), **151**, **152**.
Darling (S. T.), **9**.
Davey (J. B.), 304.
Davidson (J.), 118, 219.
Delanoë (P.), **136**, **139**, **191**, **2** , 307.
Delanoe (Mme.), 307.
Demidoff (A. P.), **189**.
Descazeaux (M. J.), **7**.
Doane (R. W.), 212.
Drake (R. H.), 205.
Duboscq (O.), 224.
Duke (H. L.), **18**, **145**.
Dupont (V.), 209, 368.

E.

Eckard (B.), **33, 34, 250.**
Edwards (F. W.), 370.
Eichborn (A.), **31.**
Ellis (A. W. M.), 109, 123.

F.

Fantham (H. B.), 93, 223.
Farrant (A. L.), 208.
Favero (F.), **20,** 288.
Ferber (F.), **24.**
Fischer (W.), 112, **250.**
Fiske (W. F.), 116.
Fletcher (W.), **271.**
Froggatt (W. W.), 302.
Franchini (G.), **175, 317, 353.**
Frosch (P.), **153.**

G.

Galli-Valerio (B.), **173.**
Gedoelst (L.), **192.**
Giemsa (G.), **286.**
Giorgio (G. di), **256.**
Gonder (R.), **167.**
Goodall (A.), **320.**
Goretti (G.), **21.**
Gough (L.H.), 371.
Gozony (L.), 225.
Graham (L. W.), **239.**
Grosso (G.), 315.

H.

Hadwen (S.), **77, 78.**
Hagemeister (W.), **251.**
Hamerton (A. E.), **14, 15, 16, 17, 32, 57, 233, 236, 321, 322, 325, 326, 327.**
Harber (A. F.), 94.
Hartley (P.), **182.**
Hartoch (O.), **45, 147, 148.**
Harvey (D.), **14, 15, 16, 17, 32, 57.**
Hata (S.), **53.**
Heckenroth (F.), **47.**
Henningfeld (F.), **156.**
Henry (A.) **185, 186, 187, 278.**
Hindle (E.), **13,** 218.
Hoepke (H.), **283.**
Hoffmann (G. L.), 107.
Holmes (J. D. E.), **67, 89,** 104, 110, 111, **354, 355.**
Howard (G. G.), **68.**
Hutchens (E.), **357.**
Hutchison (R. H.), **239.**

I.

Ismert (R.), **1.**

J.

Jack (R. W.), **333.**
Jannot (A.), **54.**
Johns (F. M.), **138.**
Johnson (J. C.), **75.**
Johnston (J. E. L.), **27, 43, 44, 264.**
Johnston (T. H.), 305.
Jonesco-Michaïesti (C.), **282.**
Jowett (W.), **81.**
Joyeux (C.), **59.**

K.

Kallert (E.), 289, 290, 291.
Kennedy (W.), 95.
King (H. H.), 115.
King (W. E.), 107, 205.
Kleine (F. K.), **34,** 112, **250.**
Knowles (R.), **343,** 362.
Knuth (P.), **11,** 124, **153.**
Kolle (W.), **45, 147, 148.**
Konrádi (D.), **177.**
Korke (V. T.), **335.**
Koselkine (P. M.), **189.**
Kozewalow (S.), 203.
Kuhn (P.), **80.**

L.

Lafont (A.), **141,** 209, 368.
Landois (F.), **283.**
Lane (C.), **347.**
Lanfranchi (A.), 210.
Launoy (L.), **50, 51, 52, 168, 265.**
Laveran (A.), **10, 39, 40, 56, 133, 137, 162, 172, 175, 243, 317, 331, 353.**
Lebailly (C.), 108.
Leese (A. S.), **131.**
Leger (A.), 300, 308, **344.**

Leger (L.), 224.
Leger (M.), **58,** 300, 308, **344.**
Leiper (R. T.), **64.**
Lemaire (G.), **55, 165, 338.**
Levaditi (C.), **41, 70, 332.**
Lévy-Bruhl (M.), **50, 52, 168, 265.**
Lewis (J. C.), 125.
Lhéritier (A.), **1, 3, 4, 55, 165.**
Lignières (J.), **230.**
Lignos (A.), 200.
Lloyd (Ll.), **274,** 303.
Lucet (A.), 226.
Lundie (A.), **157.**
Lurz (R.), **150, 246.**
Luzzani (L. N.), **71, 72.**

M.

Macfie (J. W. Scott), **27, 37, 38, 43, 44,** 215, **248, 264.**
Maciel (J.), **63, 171,** 299.
Mackie (F. P.) **336, 345.**
Maja (A.), **23.**
Manouélian (Y.), **178.**
Mansion (G.), 372.
Marchoux (E.), **49.**
Marshall (C. H.), **83.**
Martinez (I. G.), **319.**
Martini (E.), 382.
Martoglio (F.), 90.
Marullaz (M.), **137, 158, 172.**
Marzinowsky (E. I.), 373.
Mayer (M.), **160, 254.**
M'Gowan (J. P.), **196.**
Meissner (W.), 316.
Mejía (C. R.), 101.
Melli (C.), **73.**
Mellis (M. C.), **231.**
Mello (U.), **36.**
Mesnil (F.), **26, 140, 324.**
Mine (N.), 309.
Misson (L.), 287.
Minett (E. P.), **76.**
Mitra (S. N.), 126, **195.**
Mohler (J. R.), **31.**
Moldovan (J.), **61, 149,** 310.
Montfallet (D.), 127.
Montfort (F.), **46.**
Montgomery (R. E.), **181.**
Moon (V. H.), 102.
Mrowka (F.), **183.**
Mutermilch (St.), **41, 332.**
Naudin (L.), 197.

N.

Nägler (K.), **48.**
Nattan-Larrier (L.), **10.**
Nawrotzky (N. N.), **272.**
Nègre (L.), **284.**
Neiva (A.), **28.**
Neufeld (F.), 206.
Newstead (R.), 304.
Nicoll (W.), 221, 306.
Nicolle (C.), **260, 261,** 361.
Noeller (W.), **240.**
Noguchi (H.), **69.**
Nuttall (G. H. F.), **13, 78,** 91, 367.
Nyasaland Protectorate, **84.**

O.

Oehler (R.), **234, 235.**
O'Farrell (W. R.), 119, **323.**
Oliver (E. W.), 105.
Owen (G. E.), **328.**

P.

Panisset (W. L.), **281.**
Paparcone (E.), **22.**
Patton (W. S.), **337.**
Perroncito (E.), 376, 377.
Phisalix (Mme.), **342.**
Pitschugin (P. I.), 293.
Pittaluga (G.), 201, **257.**
Pixell (H. L. M.), **62.**
Pokschischewsky (N.), **180.**
Poor (D. W.), 103.
Porter (A.), 223.
Prentice (G.), **142.**
Pricolo (A.), **65, 66.**
Pringault (E.), **161, 252, 253, 259.**
Provenzale (E. F.), 90.

R.

Railliet (A.), **185, 186, 187, 278.**
Ratz (S. von), 120, **166,** 204.
Rhodesia, Southern, **85, 86.**
Richters (E.), **11.**
Ringenbach (J.), **273, 324.**
Robinson (L. E.), 118, 219.
Rocha-Lima (H. da), **254.**
Romanovitch (M.), 378.
Rondoni (P.), **42.**
Roper (R.), 374.

Rothermundt (M.), **45.**
Roubaud (E.), **141, 193,** 214.
Roudsky (D.), **243, 331.**
Row (R.), **262, 334.**
Rowley-Lawson (M.), **269, 270.**

S.

Sangiorgi (G.), **176,** 296, 297.
Sabrazès (J.), 92.
Sarrailhé (A.), **170.**
Saunders (P. T.), 379.
Scheferling, 311.
Schellhase (W.), **130.**
Schilling (C.), **42,** 211.
Shircore (J. C.), **275.**
Schokhor (N. I.), **132, 163, 164, 188, 189.**
Schürmann (W.), **45, 147, 148.**
Scordo (F.), 99
Seidelin (H.), **267.**
Sèjournant (J.), 113.
Senevet (G.), **338,**
Sergent (Ed.), **1, 2, 3, 4, 55, 165, 268, 338.**
Sergent (Et.), **338.**
Seurat (L. G.), **279,** 380.
Seyderhelm (R.), **155.**
Silva (P. da), **258.**
Simpson (J. J.), **277.**
Smith (J.), **229.**
Spagnolio (G.), 100.
Stanton (A. T.), 375.
Stefko (W.), 96.
Steinhardt (E.), 103.
Stella (V.), 90.
Stephens (J. W. W.), 294.
Stockman (S.), **228.**
Suldey (E. W.), 199.
Swift (H. F.), 109, 123.

T.

Taute (M.), **35.**
Theobald (F. V.), 117.
Thomson (J. G.), 93.
Thomson (D.), 295.
Tibadi (E.), 359.
Todd (J. L.), **341.**
Toyoda (H.), **12.**
Trautmann (O.), **6, 135.**

U.

Uganda Protectorate, **87, 88.**
Uhlenhuth (P.), **155.**
Urquhart (A. L.), **356.**

V.

Valladares (J. F.), 360.
Velu, 222.
Vincenzo (Z.), **82.**
Visentini (A.), **60.**
Vorwerk, **247.**

W.

Walker (G. K.), **154, 194.**
Watson (E. A.), **238, 280.**
Ward (A. R.), **351.**
Watson (D. P.), **233, 236, 321, 322, 325, 326, 327.**
Weck, **143.**
Wehrle (E.), 291.
Weiss (A.), 381.
Wenyon (C. M.), 121, **159, 263.**
Werner (H.), **160.**
Williams (A. J.), **79.**
Winogradoff (W. W.), **189.**
Wirth (D.), **190.**
Wolbach (S. B.), **340, 341,** 365, 366.
Wölfel (K.), 106.
Wood (W. F.), **351.**
Woosnam (R. B.), **184.**
Wragg (W. G.) **228.**
Wright (R. E.), **179.**

Y.

Yakimoff (W. L.), **132, 163, 164, 188, 189.**
Yorke (W.), **25, 29, 134, 232, 237, 244, 245.**

Z.

Ziemann (H.), 198.
Zupitza (M.), **276.**

SUBJECT INDEX OF PAPERS ABSTRACTED.

The numbers refer to the pages.

Anaplasmosis, 1–9, 61–64, 113–117, 157–159.
Bovine
A. argentinum, 115.
A. marginale
incidence, 1, 115.
immunity, 3, 114.
Equine, 63.
Ovine, 3.
Anaplasma-like bodies in the blood of mammals, 157.
See also " Reports."

Anthrax.
incidence, 41.
immunity, 198.
staining reactions, 41.

Aujeszky's Disease, 44, 151.

Babesiasis (Piroplasmosis), 1–9, 61–64, 113–117, 157–159.
Bovine
B. argentinum, 4.
B. bigeminum.
incidence, 2–4.
immunity, 3, 113, 114.
transmission, 4.
treatment. 2, 4, 117, 158.
B. divergens
immunity, 113.
Canine
cultivation, 7, 8.
identity, 5.
incidence, 158.
treatment, 158.
Equine
cultivation, 61.
incidence, 1, 4, 5, 62, 63, 158.
immunity, 62.
transmission, 4, 5, 62.
treatment, 1, 158.
Ovine, 63.

Book Review, 109.

Debab, El see *T. cazalboui.*

Dourine see *T. equiperdum.*

Drugs.
Antimony compounds, 28, 78, 80, 132, 168, 170.
Arsenic compounds, 80, 81, 83, 168.
Arsenobenzol, 172.
Atoxyl, 76, 79.

Drugs—*cont.*
Bromine, 27.
Diaminoarsenobenzene derivative, 171.
Iodine, 27.
Neosalvarsan, 29.
Osmic acid, 27.
Salvarsan, 79, 82, 132, 133.
Silver Salts, 80, 81.
" 606 " 26, 52.

Epizootic Lymphangitis.
nature of organism, 52, 152.

False Rabies see **Aujeszky's Disease.**

Flies.
Methods of catching and control, 146, 147.
Chrysops
transmission of *T. equinum*, 19.
Gedoelstia cristata, 107.
Glossina
G. brevipalpis, 159.
G. fusca, 100.
G. morsitans
development of *T. pecorum* in, 12.
— *T. brucei*, Zululand, 121.
— trypanosome causing human disease in Nyasaland, 123.
connection with game in Rhodesia, 174.
G. palpalis
transmission of *T. rhodesiense*, 22.
Haematopota
transmission of trypanosomes, 12.
Kirkia
K. blanchardi, 106.
K. surcoufi, 106, 107.
Lyperosia minuta
transmission of horse sickness, 51.
Oestruus variolosus, 107.
Stomoxys nigra
transmission of *T. nigeriense*, 25.
Tabanidae
transmission of trypanosomes, 12.

Helminthiasis, 39–40, 101–106, 149 184–185.,
Algeria, parasite of flamingo, 149.
Annam, Amphistomes of Rummants, 149.
Asia, parasites of Elephant, 101, 102.
—, trematodes of dog, 39.
Austria, microfilaria of horse, 105.
Philippines, parasite of pig, 184.
Tripoli, parasite of camel, 39, 40.
Turkestan, microfilariasis, 104.
Parasites of
camel, 39, 40, 104, 140.
cattle, 104, 149.
dog, 39, 104.
elephant, 101, 102.
flamingo, 149.
Anoplocephala manubriata, 101.
Ascaris lonchoptera, 102.
Bathmostomum sangeri 102.
Bunostomum foliatum, 102.
Centrocestus cuspidatus, 39.
Choniangium, n. gen. 102.
— *epistomum*, 102.
Clonorchis endemicus, 39.
Cylicostomum falciferum, 102.
— *pileatum*, n. sp. 102.
— *sipunculiforme*, 102.
Echinococcus veterinorum, 101.
Evansia renniei, 102.
Fasciola jacksoni, 101.
Filaria haematica cameli, 39.
— of *Agama colonorum*, 106.
Gastrothylax cobboldi, 149.
— *cruminifer*, 149.
— *elongatus*, 149.
— *minutus*, 149.
Grammocephalus clathratus, 102.
Heterophyes heterophyes, 39.
Homalogaster paloniae, 149.
— *philippinensis*, 149.
— *poirieri*, 149.
microfilaria of *Agama colonorum*, 106.
Opisthorchis noverca, 39.
Paragonimus westermanii, 39.
Paranphistomum bathycotyle, 149.
— *explanatum*, 149.
— *fraternum*, 149.
— *orthocoelium*, 149.
— *papillosum*, 149.
Pfenderius papillatus, 101.
Pseudodiscus hawkesii, 101.
Schistosoma japonicum, 39.
Stephanurus dentatus (*Sclerostoma pinguicola*), 184.
Strongylus addidictus, 102.
— *instabilis*, 40.
Tropidocerca coccinea, 149.

Helminthiasis—*cont.*
Watsonius ornatus, 101.
Yokogawa yokogawa, 39.

Horse Sickness.
transmission, 51.

Leishmaniasis.
Canine
incidence, 33, 86, 87, 139, 140, 141.
lesions, 87.
pathogenicity, 33, 140, 141.
transmission, 139.
Human
cultivation, 86.
incidence, 175, 176, 178.
transmission, 177.
L. donovani
pathogenicity of cultures, 174.
L. tropica
pathogenicity of cultures, 174.
behaviour in dog flea, 176.

Leucocytozoa.
Leucocytogregarina cuniculi, 94.
Leucocytozoon ziemanni
development, 37.

Malaria.
of duiker, 34.
— flying fox, 183, 184.
— man, 144, 145.
— mole, 37.
— monkey, 35, 143, 146.
— pigeon.
Haemoproteus columbae
development, 180.
immunity, 144.

Miscellaneous.
Acarus, endoparasitic in lung of monkey, 151.
Anaemia, infectious equine, 188.
Bursati, 195.
Dysentery bacillary in monkey, 151.
Flagellates of invertebrates, 193.
Haemogregarina boodoni, 180.
Haematozoa from Congo, 145.
Mycosis pulmonary of Ostrich, 52.
Pasteurella of Indian Elephant, 108.
Staining with Giemsa, 153.
Yellow fever, monkey as reservoir, 152.

Nagana, see *T. brucei*.

Nuttalliasis, see Babesiasis.

Pleuropneumonia
goat, 108.

Protozoology, (see also "Malaria" and "Miscellaneous")
Amoeba of macacus, 93.

Protozoology—*cont.*
Flagellates of *Ctenocephalus canis*, 93.
Haemamoeba of *Liothrix luteus*, 92.
Toxoplasma of —, 92.
Smithia talpae, 92.

Rabies, 42–44, 94–96.
cultivation, 42.
diagnosis, 43.
histology, 95.
immunity, 96.

Rabies False see **Aujeszky's Disease.**

Recent Literature see **separate Index to References**, 213.

Reports.
Nyasaland, 53.
Rhodesia, 53, 54, 201.
Uganda, 55, 200.

Rinderpest.
Asiatic, 99.
blood, examination of, 45.
cultivation, 185, 186.
immunity, 97.
transmission, 187.

Sleeping Sickness, see *T. gambiense*, *T. rhodesiense*, *T. nigeriense.*

Spirochaetosis, 30–33, 88–89, 142–143, 178–179.
Avian
cultivation, 143.
immunity, 32, 88.
incidence, 88, 142.
lesions, 32, 142.
transmission, 30.
Human
cultivation, 33.
development, 178.
filtrability, 179.
Other species
goat, 148.
pig (swine fever), 89.
sheep, 148.

Theileriasis, 9–10.

Ticks.
Amblyomma cajannense, 5.
Argas persicus, 30.
Boophilus annulatus, 5.
Dermacentor nitens, 5.
Margaropus annulatus, var. *australis*, 4.
Margaropus microplus, 115.
Ornithodorus moubata, 178.
Rhipicephalus bursa, 4.

Tick Paralysis.
in man and sheep, 48.
— dog, 50.

Toxoplasmosis, 38, 90–92.
Canine
identity, 38.
pathogenicity, 38.
Toxoplasma gondii
biology, 90.
cultivation, 38.
multiplication, 38.
pathogenicity, 38, 90.
transmission, 92.

Trypanosomiasis, 10–30, 64–85, 117–138, 159–174.
General (see also individual trypanosomes).
biology, 23, 72.
cultivation, 17, 135.
differentiation, 117.
diagnosis, 27.
drugs, effects of, 27, 28, 76, 78–83, 132, 133, 168.
electricity, effects of, 83.
fixation abscess, 76.
immunity, 26, 27, 69.
isolation, 84.
morphology, 19.
staining, 153.
transmission, mechanical, 167.
birds, 84.
camel, 64, 66, 168.
cattle, 12, 70.
dog, 70.
dromedary, 15.
game, 72, 75.
goat, 70.
horse, 12, 19, 70.
man, 13, 21, 71, 72, 123, 159, 163.
monkey, 10.
mule, 48.
pig, 70.
sheep, 70.
T. americanum
cultivation, 70.
T. annamense, 20.
T. brucei
development, 121.
identity, 119..
immunity, 69.
incidence, 165.
morphology, 119, 122, 123.
pathogenicity, 120.
transmission, experimental, 16.
T. brucei (Uganda strain).
identity, 18.
T. caprae
incidence, 165, 166.
T. cazalboui
incidence, 70, 148.
pathogenicity, 68.
T. congolense
identity, 67.
T. dimorphon
incidence, 70, 148.
pathogenicity, 68, 134.

Trypanosomiasis—*cont.*
T. duttoni
pathogenicity, 26, 131.
T. equiperdum
biology, 19.
diagnosis, 20, 21, 126.
identity, 20.
incidence, 126.
T. evansi
treatment, 168.
T. gambiense
development, 135.
identity, 164.
incidence, 72.
pathogenicity, 24.
transmission, 72.
T. ingens
incidence, 17, 166.
T. lewisi
morphology, 130.
pathogenicity, 130, 131.
transmission, 130.
T. nanum
identity, 67.
T. nigeriense
development, 25.
morphology, 25.
pathogenicity, 25, 133.
T. pecaudi
incidence, 70, 148.
T. pecorum
development, 159.
incidence, 14, 23, 165, 166.
Trypanosomiasis—*cont.*
pathogenicity, 12.
transmission, 12.
T. rhodesiense
development, 135.
identity, 125, 164.
incidence, 23, 72, 165.
transmission, 22, 72.
T. simiae
development, 11.
incidence, 165, 166.
pathogenicity, 10.
transmission, 10.
T. soudanense, 66.
T. uniforme
incidence, 14.
T. vespertilionis
pathogenicity, 137..
transmission, 137.
T. vivax
incidence, 14, 23.
pathogenicity, 18.
Schizotrypanum cruzi
biology, 30, 138.
morphology, 137.
multiplication, 137, 138.
pathogenicity, 30, 137.

Undulant Fever.
Antibodies in cows' milk, 199.
effect of "606" on, 52.
virulence, 52.

INDEX TO RECENT LITERATURE.

The numbers refer to the pages.

Anaplasmosis, 203.
Aujeszky's Disease, 110, 155.
Babesiasis, 58, 110, 154, 203.
Epizootic Lymphangitis, 58.
Flies, 59, 111, 155, 204.
Foot and Mouth Disease, 154.
Helminths, 58, 110, 112, 155, 204.
Leishmaniasis, 58, 110, 154.
Malaria, 154.
Mosquitoes, 60, 111.
Protozoa, 60, 112, 155, 204.
Rabies, 58, 110, 203.
Rinderpest, 59.
Snake Bite, 203.
Spirochaetosis, 59, 111, 203.
Ticks, 60, 112.
Tick Paralysis, 203.
Trypanosomiasis, 59, 111, 155, 203.
Unclassed, 60, 112, 156.
Undulant Fever, 59.